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ESTIMATED PUBLIC BENEFITS OF IMPLEMENTING THE PROPOSED REVISIONS TO REGULATION 308

VOLUME II – APPENDICES A TO C

JULY 1990



Ontario

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Jim Bradley, Minister/ministre

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ESTIMATED PUBLIC BENEFITS OF IMPLEMENTING
THE PROPOSED REVISIONS TO REGULATION 308
VOLUME II - APPENDICES A TO C

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and
Monenco Consultants Ltd.

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APPENDIX A

APPENDIX A

Economic Valuation of
Benefits

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A.1 INTRODUCTION

One aspect of this project is to estimate an economic value for benefits related to changes in air pollution, or air quality, stemming from proposed revisions to Regulation 308.

This appendix introduces the economic concepts and methods used to estimate the value of changes in social welfare due to changes in air quality. Additional discussions may be found in Freeman (1979), and Rowe and Chestnut (1983) among others. Detailed discussions of these concepts, measures, and methods as applied to specific impact categories may be found in Chestnut and Violette (1985), and Violette and Chestnut (1983) for valuing health impacts, in Rowe et al. (1984) for valuing agricultural impacts, in Rowe and Chestnut (1983) for valuing visibility impacts, and in Manuel et al. (1983) for valuing materials damage and soiling impacts.

Throughout this appendix changes in air quality are said to result in "benefits" (or to have a positive value) if the well-being of economic agents is improved, and to result in "damage" (or to have a negative value) if the well-being of economic agents is decreased. In this sense, benefits and damage are the inverse of one another. However, the term damage is frequently used to refer to physical impacts, rather than changes in well-being. To avoid possible confusion between "damage" in a physical sense and "damage" as a reduction in well-being, the term "benefits" will be used to cover all changes in well-being. Thus, benefits can be either positive or negative (ie reductions in well-being).

A.2 ECONOMIC AGENTS

The economic concept of value is the well-being derived from the consumption, productive use, or knowledge of the existence of a particular good or service. Then, any change in the level of consumption has value as long as someone's well-being is enhanced or diminished. Economists refer to the level of well-being of consumers as their level of utility. The well-being of a firm producing goods and services is usually related to the return on investment or profits (a more exact economic measure is defined below). Economic models of the behaviour of producers and consumers assume that they attempt to maximize their well-being, subject to constraints. In this sense producers and consumers are "economic agents" that attempt to maximize their well-being.

A.3 CONCEPTS OF ECONOMIC VALUE

Goods and services bought and sold in the marketplace are easily recognized as having economic value because individuals (including firms) spend money to purchase them at market prices and forego purchasing other goods and services that could have increased their well-being. Many environmental and natural resources, such as air quality, are not bought or sold in the marketplace and, therefore, do not have explicit prices. This does not mean that they do not have economic value. People change their recreation patterns, move their residences, or experience changes in their health and well-being due to air quality characteristics. Similarly, firms often choose locations based upon the availability of unpriced environmental amenities such as human health benefits to employees and visual aesthetics.

Values (estimated prices) of environmental and natural resources are difficult to determine. But techniques have been developed to estimate the value of a change to a resource such as air quality. The total value of a change to an environmental or natural resource is often discussed in terms of three components:

- . User values held by consumers and producers;
- . Option prices held by potential users; and
- . Preservation, or non-use, values held by any economic agent.

A.3.1 Use Values

"Use values" are held by individuals who engage in an activity involving, or affected by, air quality. This may entail the use of air through a consumption activity, such as using good air quality to breathe or to view distant scenery, or using good air quality as a productive input to grow crops.

A.3.2 Option Prices

When future use is uncertain, an "option price" is part of the change in total economic value. This is the present value of preserving the option of using the resource in the future. It reflects the expected future use value plus any risk premiums (positive or negative) related to uncertain existence and demand for the change in the resource (See Freeman, 1988, 1985; and Smith, 1987a, b, 1983 for further discussion of this concept). The option price is the correct measure of a change in resource quality that has future impacts, but option price may be difficult to measure. The option price can be approximated by estimating the probability of future use and future use values, then discounting these values to the present (See Lind et al., 1983; Sandler and Smith, 1976, 1977, 1982 and Schulze and Kneese, 1981 for discussion of discount rates).

A.3.3 Preservation Values

Beyond current and potential future use of a resource, individuals may have other motives, and therefore values, for the preservation of resources. Such values, referred to as "preservation or non-use values" may be associated with providing the resource for others to use now and/or in the future ("bequest", "altruism", or "vicarious consumption" values), or simply for the sake of preserving the resource in the same state of being ("stewardship" or "pure existence" values). The theoretical foundations for, and empirical estimation of, preservation values is developing, and empirical estimation of these values is of uncertain accuracy. (See Schulze et al., 1981; McConnell, 1982; Randall and Stoll, 1982; Fisher and Raucher, 1983; Boyle and Bishop, 1985). Nevertheless, empirical work to date suggests that option prices and preservation values may be a substantial share of the total change in value for a resource.

Pioneering studies that have estimated option and other preservation values (Smith, 1983a, 1983b; Freeman, 1984; Fisher and Raucher, 1983) have found that both users and non-users hold significant option and other preservation values for the protection of visibility and other natural resources. Considering the significant number of non-users who may hold these values, the aggregate of option prices and other preservation values held by all individuals may exceed the aggregate of use values by users for changes in many natural resources and their related impacts.

Due to the uncertainty inherent in estimates of option and other preservation values, and limited literature upon which to base estimates, these values have generally been excluded from the

calculations of economic benefits. As a result, benefit estimates may substantially understate total benefits.

A.4 MEASURES OF ECONOMIC VALUE

Economics attempts to quantify changes in well-being using monetary measures. In general, the monetary measure of the change in an individual's well-being due to a change in air quality is the change in income that yields the same or offsetting change in the individual economic agent's well-being as does the change in air quality or the effects of the change in air quality. Separate measures are defined for consumers and producers. Total social economic benefits due air quality improvements is then defined as the aggregate of all benefits to individual consumers and producers.

Three measures are often used in economic analyses of changes to consumer well-being:

- . willingness-to-pay (WTP);
- . expenditures; and
- . consumer's surplus.

A.4.1 Consumer's Surplus

Willingness to pay (WTP) is the maximum amount an individual is willing to pay for a good or service. This is its maximum monetary value to the individual. In many cases, price is an accurate reflection of the maximum WTP for additional units of a good or service. "Expenditures" are the amount a consumer spends for a good or service (price times quantity), which in a situation of free choice is never greater than his maximum WTP for all units consumed. However, an individual may be fortunate

enough to spend less than his maximum WTP. This difference between maximum WTP and actual expenditures is referred to as consumer's surplus.

The change in consumer's surplus is used as the economic measure of the change in utility due to change in air quality or the effects of air quality. Because air quality is a natural resource, expenditures are seldom required for use or preservation of air quality. Hence changes in WTP and consumer's surplus are usually equivalent.

A.4.2 Ordinary Consumer's Surplus

Demand curves are used to measure maximum WTP, expenditures, and what is called "ordinary" consumer's surplus. A demand curve is the relationship between quantity of a good or service demanded and the maximum WTP per unit in terms of price. A demand curve is typically downward sloping because each additional unit consumed yields a smaller increase in utility; therefore the individual's WTP for additional units also decreases. The maximum WTP for each additional unit of a good is represented by the corresponding point on the WTP, or price, axis. For example, in Exhibit A.1, the WTP for an additional unit of X at level Q_1 is P_1 . If an individual were charged price P_1 for each of the Q_1 units, expenditures would equal the area under the demand curve represented by the rectangle (OQ_1DP_1). Ordinary consumer's surplus (OCS) equals the difference between the area under the ordinary demand curve up to the quantity consumed and expenditures (P_1DA).

Expenditures and consumer's surplus may change due to changes in prices and available quantities of a good. For example, if the market price falls from P_1 to P_2 , the quantity the consumer demands increases from Q_1 to Q_2 and consumer's surplus increases by (P_1P_2ED). The consumer benefits by paying less for the units

EXHIBIT A - 1

Demand for a Market Good

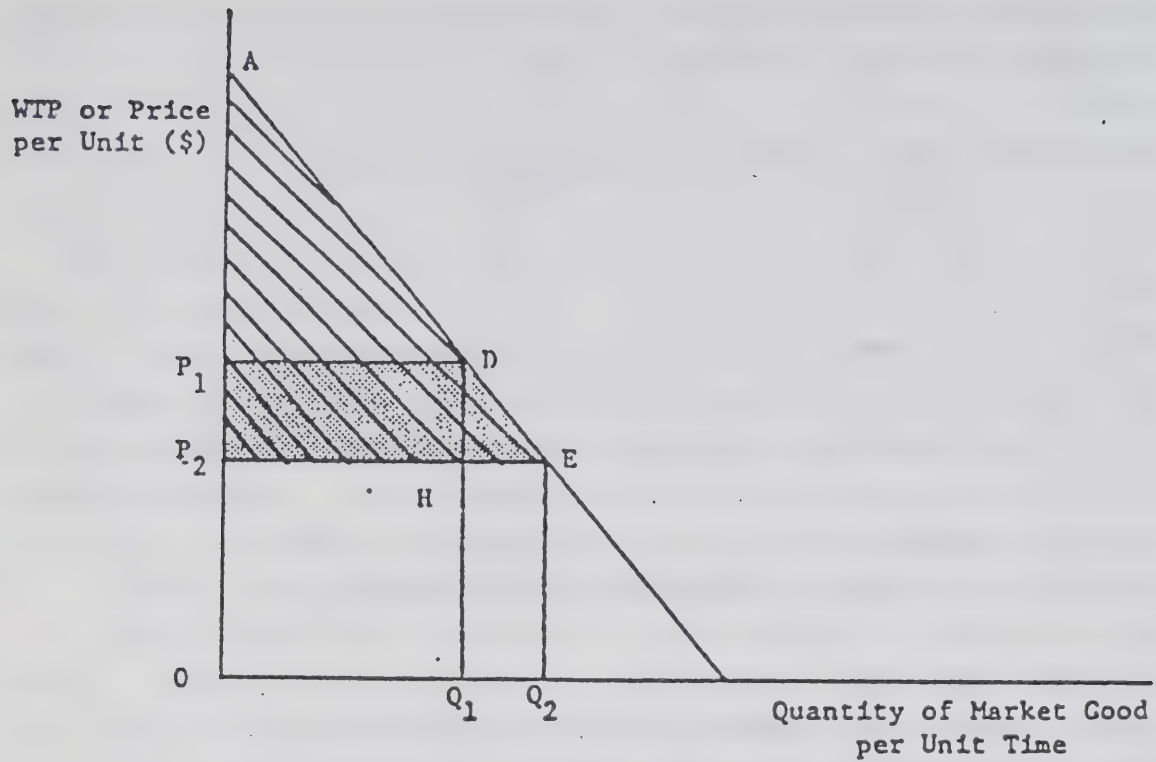
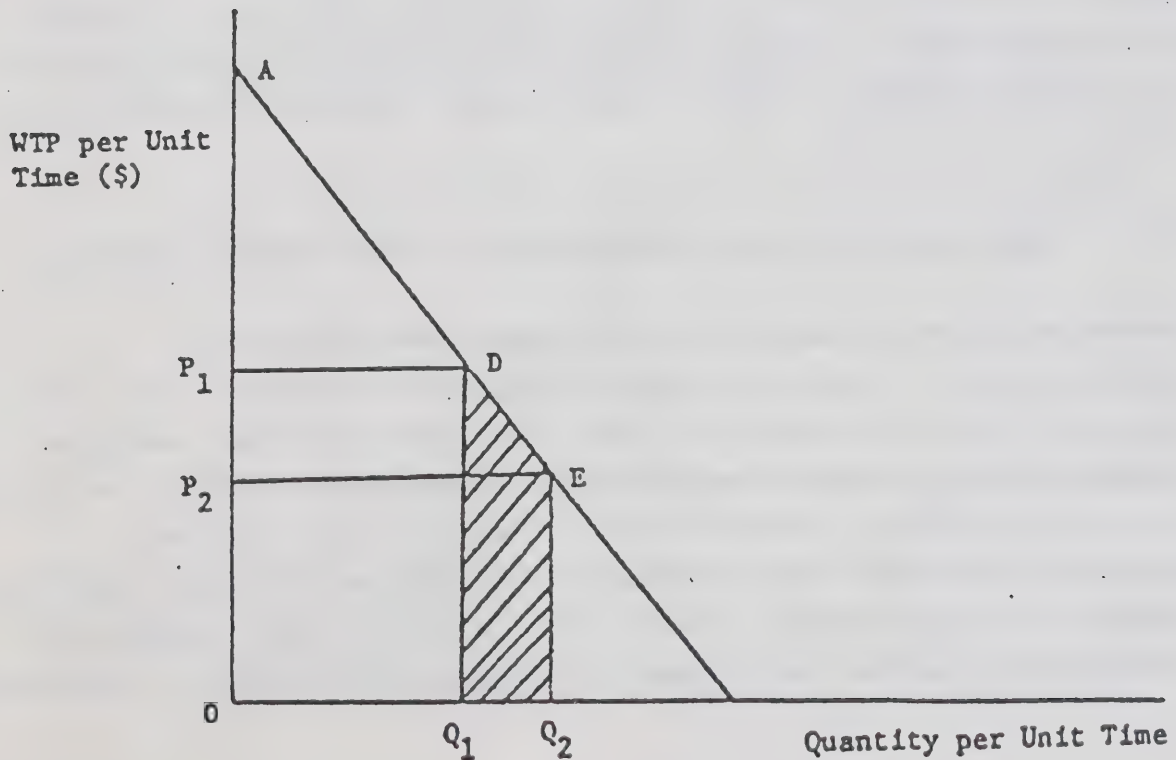


EXHIBIT A - 2

Demand for a Non-Market Good:
The Effect on Consumer's Surplus of a Reduction
in the Availability of Resource Q



previously consumed (P_1DHP_2) and by paying less than his maximum WTP (DEH) for the additional units consumed.

A.4.3 Estimating Ordinary Consumer's Surplus

In many cases changes in air quality affect the price and availability of market goods. Then the demand curve for these market goods can be used to infer the economic loss to consumers. For example, decreased air pollution may result in lower materials replacement or cleaning costs and therefore in reduced prices to produce and maintain these goods. In Exhibit A.1, this could be represented by a price decrease from P_1 to P_2 with a resulting consumer's surplus gain of (P_1DEP_2).

Many services that embody air quality are not traded. In these cases, there is no market price that can be used to estimate the consumer's surplus due to a change in air quality. When this occurs, expenditures are zero and maximum WTP, or the entire area under the demand curve up to the available quantity, equals ordinary consumer's surplus. For example, in Exhibit A.2, if Q_2 units of a good, such as visual range, are available at zero price, consumer's surplus is the area (OAE Q_2). If air quality degradation reduces visual range to Q_1 , consumer's surplus is reduced by (Q_1Q_2ED).

A.4.4 Theoretically Correct Measures of Consumer's Surplus

The "ordinary" consumer's surplus measure is in many circumstances a very good approximation of theoretically correct consumer's surplus measure. But it is not an exact measure of consumer's surplus because utility is not held constant at points along the ordinary demand curve. Theoretically correct consumer's surplus measures have been defined (Hicks, 1944) and refined by many authors. Recall that the economic measure of change in value is the change in income that results in the same

or offsetting change in utility. The consumer's surplus measure will differ whether one is measuring the change in income that provides the same change in utility, or an offsetting change in utility, i.e., whether the initial level of utility is to be used as the reference point or the new level of utility is to be used as the reference point.

Thus, there are two theoretically correct measures of consumer's surplus, depending upon the reference point. Four approaches to estimating these measures of consumer's surplus have been developed.

- . Compensating Surplus and Compensating Variation Measures, which keep the individual at the initial level of utility.
 1. The maximum willingness to pay (WTP) for an improvement in natural resource conditions.
 2. The minimum willingness to accept compensation (WTA) for a reduction in natural resource conditions.
- . Equivalent Surplus and Equivalent Variation Measures, which keep the individual at the new level of utility.
 3. The WTP to avoid a reduction in natural resource conditions.
 4. The WTA to forego an improvement in natural resource conditions.

A.4.5 Correspondence Among Measures of Consumer's Surplus

There is strong theoretical evidence that the ordinary consumer's surplus measure and the four refined measures should be very similar under many circumstances (Willig, 1976; Randall and

Stoll, 1980; Bockstael and McConnell, 1980). The conditions when the measures would, according to economic theory, be substantially different are when the expenditures or values for a good or service are a large component of income, the changes in the good being valued are very large, and the income elasticity of WTP is large.

Applied work has found large differences in empirical estimates of these measures even when the theoretical conditions suggest they should be quite similar. It is currently thought that these empirical discrepancies are more likely the artifact of application issues, such as property rights and ethical acceptances, than the underlying theoretical foundations (Rowe and Blank, 1981; Gregory, 1983; Weinstein and Quinn, 1983).

Theoretically, the selection of the correct measure is generally straightforward, but because of the large variations in empirical estimates this becomes an important concern in practice. The WTP measures (#1 and #3 above) have received greater acceptance by the profession, as the results using these approaches have been more stable and have had lower variances.

A.4.6 Producer's Surplus

The change in well-being for producers of goods and services due to a change in air quality is measured as the change in producer's surplus resulting from this change in the air quality. Producers of goods and services often utilize air quality as a productive input. If air quality improvement reduces their costs of producing the same goods and services, the producers may be better off.

For example, reduced air pollution may raise agricultural yields, or equivalently, lower production costs. A typical supply curve, as depicted in Exhibit A.4, represents the marginal cost of

Demand for Non-Market Good:
The Effect on Consumer's Surplus of a Reduction
of the Quality of the Resource Q

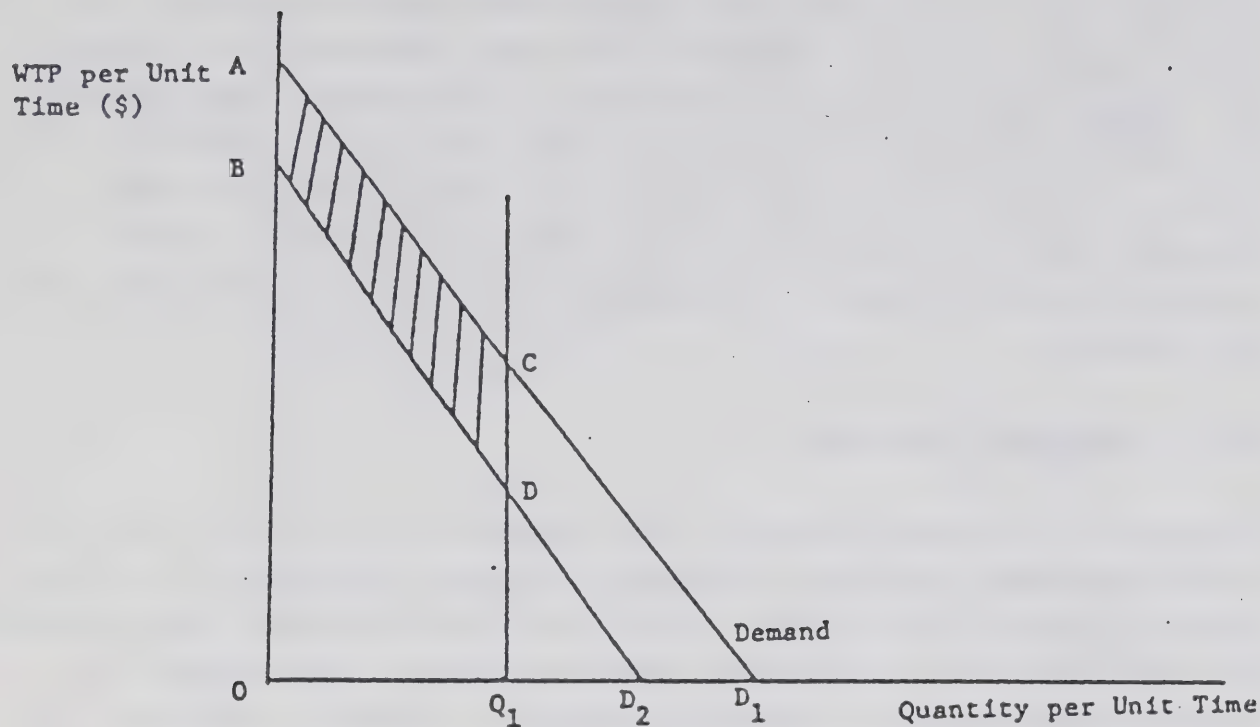
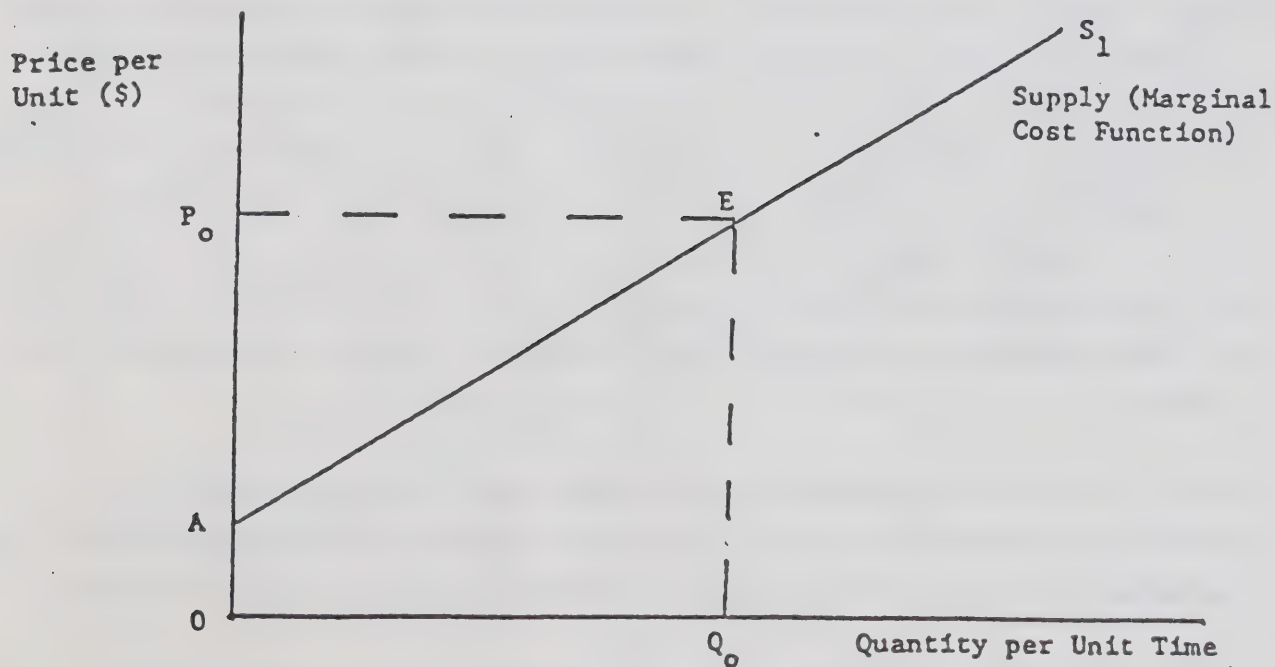


EXHIBIT A - 4

Supply of a Market Good



production and equals the minimum monetary compensation a producer will accept and still supply the good or service. At price P_0 , Q_0 units are supplied and total revenues are (OP_0EQ_0) . The total marginal cost of producing Q_0 is $(OAEQ)$. This leaves an excess of (AP_0E) as "producer's surplus" to be used as a return to fixed factors of production. It is the difference between what the producer receives and the minimum he must receive to be willing to supply the additional units of the good or service.

A.4.7 Economic Surplus

The sum of consumer's surplus and producer's surplus is referred to as "economic surplus". Economic surplus, illustrated as the shaded area in Exhibit A.5, is the difference between the maximum WTP for each unit of the good and the minimum willingness to accept compensation to produce additional units of the good. It represents the net benefit to society from the transactions between the producers and consumers.

Air quality, is not supplied by a firm, rather it is available at fixed levels at zero price. A change in the of air quality only affects consumers, and economic surplus equals the consumer's surplus, as illustrated as the shaded area in Exhibit A.3. If air quality degradation reduces the quality of an input, such as recreation at a forest where ozone exposure has resulted in foliar injury, then the maximum willingness to pay for each unit of the resource (forest visits) may shift downward from D_1 to D_2 and the change in consumer's and economic surplus equals the area $(BACD)$.

If air quality degradation increases the marginal cost of producing a good or service, such as agricultural crops, then the minimum price at which the producer will be willing to supply

Economic Surplus

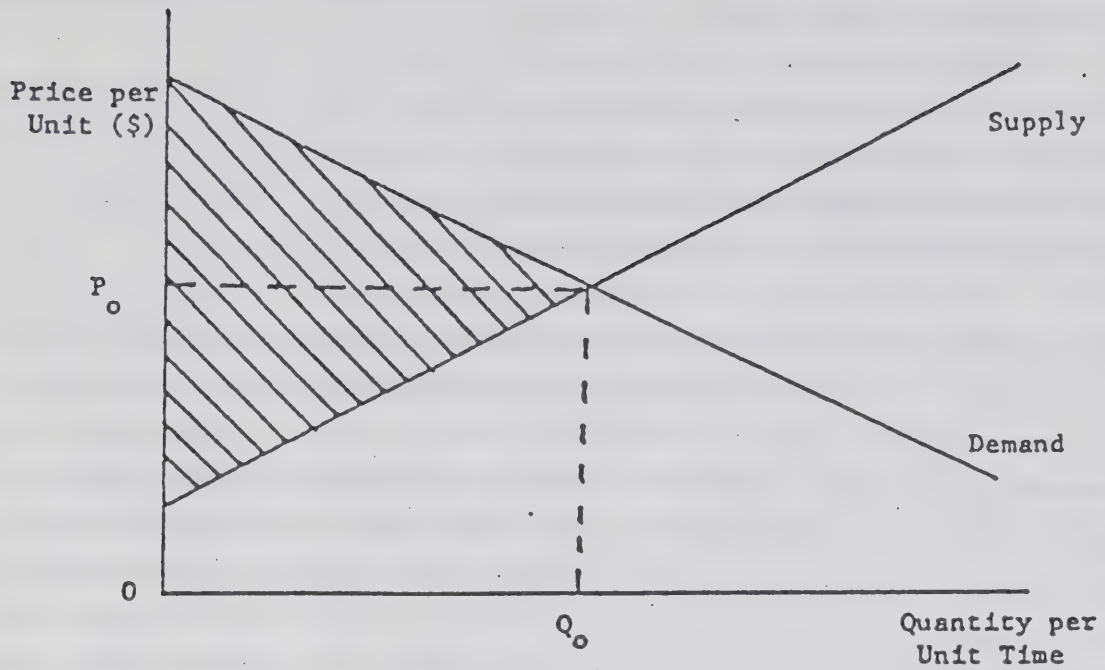
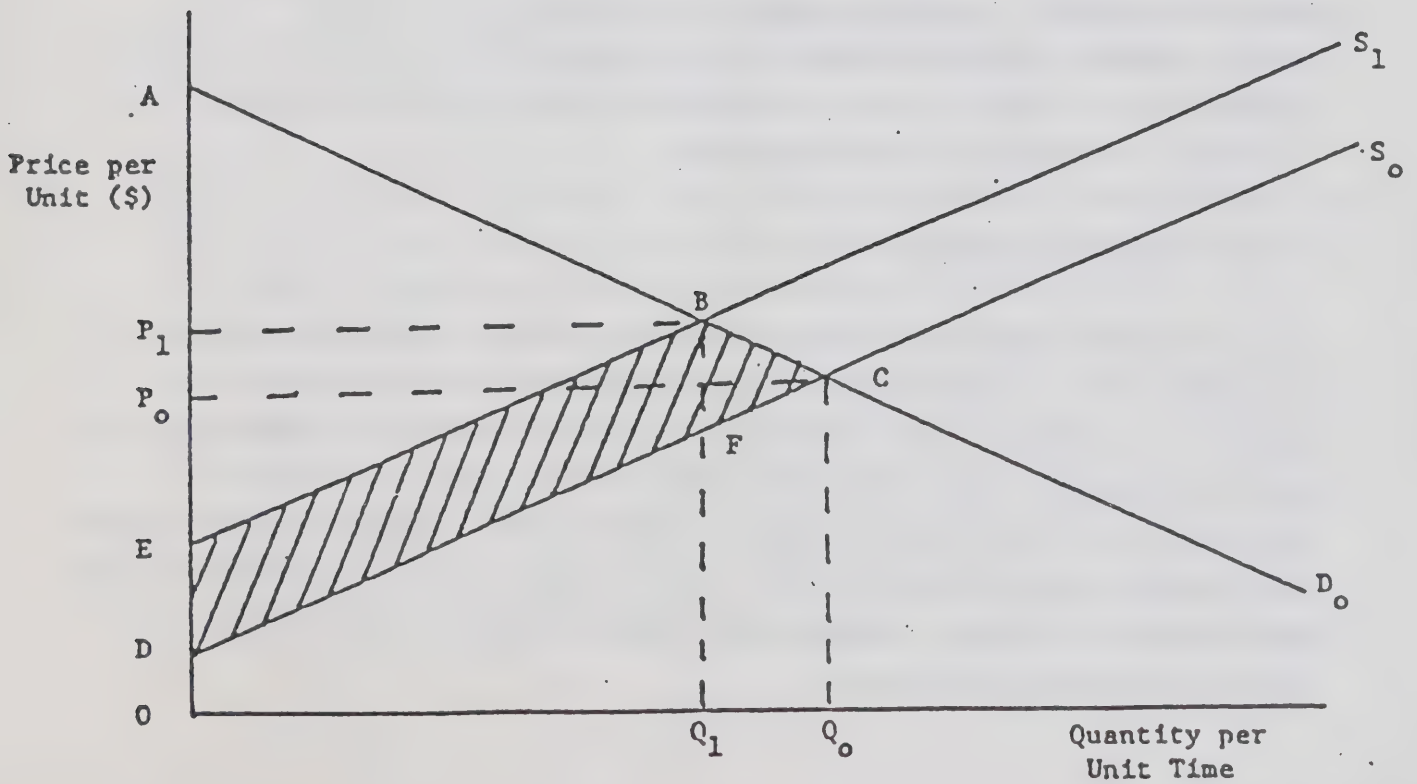


EXHIBIT A - 6

Effects of Air Quality Degradation Upon Economic Surplus



each additional unit of output will increase. This is illustrated by the shift in supply from S_0 to S_1 in Exhibit A.6.

In this case the market clearing price, the price where quantity demanded equals quantity supplied, increases from P_0 to P_1 and the quantity traded decreases from Q_0 to Q_1 . The change in economic surplus is the area DEBC.

This represents a real loss in the sense that consumers must spend more for each of the Q_1 units of crops still consumed, leaving less for expenditures on other goods. Producers also lose as each unit produces lower producer's surplus, providing a lower return on investment. The reduction in expenditures to produce and consume $Q_0 - Q_1$ units of the good, represented by the area FCQ_0Q_1 , is not an economic loss because these funds are available for production and consumption of other goods and services.

Although not immediately apparent graphically, the change in economic surplus equals the sum of the changes in consumer's surplus (P_1BCP_0) and the change in producer's surplus ($P_0CD - P_1BE$). These illustrations are, of course for an idealized and generalized market product situation, and may need modification to apply to specific circumstances.

A.4.8 Total Benefits

Total benefits from a change in air quality is the sum of all benefits to all individuals. This includes benefits for consumers and producers for all impact categories. Because many individuals may have been affected in the past, or may be affected into the future, their benefits are generally converted to present dollars using a discount rate. This report estimates only annual values. Future use values are not discounted and added to present impacts.

A.5 METHODS FOR ESTIMATING ECONOMIC VALUE

A variety of methods are available to estimate the economic value of air quality changes. This section highlights these approaches and their major strengths and weaknesses. Sources for additional reading are provided for each method. The methods can be grouped into the following classifications:

- . Market, or actual valuation, methods -- Methods that infer values for changes in air quality based upon actual market data and related behaviour.
- . Non-Market, or contingent valuation, methods -- Methods that estimate values through the use of contingent, or hypothetical, markets in order to reveal what expenditures and behaviour would be if those markets were to exist.
- . Other implicit valuation approaches -- Other methods that may be used to value changes in resources, such as the analysis of voting results.

Market valuation methods rely on the observed expenditures and behaviour that individuals undertake in response to changes in air quality or the effects of changes in air quality. Their major limitation is the limited number of issues they can address due to the availability and quality of data, especially for newly emerging issues or unique sites. Also, it is often difficult to isolate the environmental values that are reflected in the markets. For example, the estimated values from urban air quality property value studies are difficult to separate into health effects, visual aesthetics effects, or materials damage.

The strength of the contingent valuation methods is that they may be applied to estimate a wide variety of values for many different air quality impacts, including problems that have not yet been experienced. The major limitation is that they rely upon predicted behaviour within the context of hypothetical situations and, consequently, may be subject to inaccuracy.

A.6 ACTUAL VALUATION METHODS

A.6.1 Simple Damage Functions

One of the earliest approaches to estimating an economic measure of environmental damages was to measure, or estimate, physical damages and multiply them by a fixed market price. For example, one could estimate the effects on agricultural yields, forest yields, or materials soiling from air pollution and then multiply these physical damage estimates by existing acreage or materials inventory and market prices to obtain a dollar damage estimate.

The principal strength of this approach is that it is easy to apply to get a first order, or crude, estimate of damages. The principal weakness is that it does not estimate a theoretically correct measure of damages because the averting and mitigating behaviour of economic agents is ignored. For example, if air pollution affects one type of material, producers may simply substitute another suitable material with only a slight increase in costs. Damage function approaches generally overstate damages of environmental pollution and understate benefits of pollution clean-up. Because it is not theoretically valid, this methodology is not used in this analysis unless estimates from better estimation methods are not available or the incremental costs of obtaining preferred estimates are not merited.

A.6.2 Hedonic Approaches

Hedonic approaches attempt to define markets in which a good or service is traded and where the quality or quantity of the environmental resource could be considered a characteristic of that good or service. By examining how the price of the traded good or service is affected by the quality or quantity of the environmental resource, an implicit price for that resource can be inferred (Rosen, 1974).

This approach presupposes that individuals respond in a predictable manner to the environmental conditions they encounter in places where they live, work and recreate. For example, it is expected that individuals prefer to live and recreate in locations where the air is clean, and prefer to work in locations where the risks to health are low, and that such preferences will be revealed by how much they are willing to pay for property, or are willing to accept in wage and salary compensation. Property markets and labour markets are the ones most commonly examined when using the hedonic approach.

Applications of this approach to environmental resource valuation issues are common, including several dozen property value studies for air pollution in urban areas (reviewed in Rowe and Chestnut, 1982; and Freeman, 1979) as well as one for recreation areas (Willman, 1980). Wage and salary studies include those by Rosen (1979) and Cropper and Arriaga-Salinas (1980).

The strength of the hedonic approach is that it uses actual market data that reflect what people have actually spent to obtain various levels of air quality or to reduce the impacts of air pollution. The usefulness of hedonic approaches is restricted by the lack of market goods or adequate market data that may be associated with air quality, or air pollution

effects, and by the inability to separate benefit estimates by effects or value categories.

Other important limitations concern the statistical procedures that are employed to estimate the implicit price. For example, the determination of the appropriate functional forms, how the market should be segmented, and the appropriate method to differentiate between demand and supply effects is problematic. Finally, the approach assumes that individuals in the market being examined are aware of the environmental quality level that is being examined and have had time to adjust their behaviour accordingly. For example, a study relating property values to the level of toxic air pollutants would lead to spurious value estimates if individuals had no knowledge, or possessed inaccurate perceptions, concerning the levels of these pollutants at alternative locations.

A.6.3 Travel Cost Approaches

The travel cost method, using actual travel behaviour as suggested by Hotelling and initially developed and applied by Clawson (1959) and Clawson and Knetsch (1966), has been used extensively to estimate the demand for recreational activities. This approach recognizes that to use the services of a site, users not only incur expenses for entry fees and equipment, but must also get to the site.

The cost, or price, to an individual of using the services at a recreation site will vary according to the travel time and expenses incurred getting to the site. As the characteristics of the site (such as air quality or site characteristics affected by air pollution) change, the number, frequency, duration, and costs of the trips will also change, reflecting a change in demand and consumer surplus. The travel cost approach has been used to value the existence of recreational sites. However, recent

research has initiated efforts to use variations of the approach to value changes in characteristics of recreational sites. These include Desvousges (1985) and Johnson and Haspel (1982). A recent thorough review of the state-of-the-art may be found in Bockstael et al. (1985).

The travel cost approach is defended on the grounds of its use of observed behaviour. However, there are still many problems with its application, including how to deal with the value of time in travel and at the site (McConnell and Strand, 1981; Willman, 1980; Cesario, 1976), handling of substitution between sites (Morey and Rowe, 1985), separating travel expenses in multi-purpose and multi-destination trips (Johnson and Haspel, 1982), capacity problems (Cicchetti and Smith, 1973), and statistical issues concerning functional forms.

A.6.4 Supply and Demand and Programming Models

Where goods and services exist that embody environmental resources, the economic surplus measure of changes in these resources can often be estimated by using programming models of the economic agent's behaviour combined with supply and demand models of market interactions. In this type of approach, physical damage functions that relate environmental pollutants to the quality and quantity of a market good, such as crop or forest yields are estimated. Next, the optimizing behaviour of the producer of the affected good is modeled with the programming model. For example, as pollution increases, a farmer may switch to more tolerant crops, increase fertilizer use, increase acreage, or undertake any number of cost effective strategies to mitigate pollutant impacts. The yield effects, combined with the farmer's actions, serve to alter the market supply function, which in combination with the market demand function, alters the equilibrium price, quantity, and economic surplus (Heck et al., 1983).

The strength of this approach is the defensibility of an analysis that relies upon actual market behaviour. Unfortunately, the data requirements are often overwhelming. The underlying specification problems in identifying the programming model objectives and constraints, and in estimating the supply and demand models compound the difficulties of using this approach.

A.6.5 Expenditure Functions

This approach attempts to value changes in environmental quality by measuring how changes in dollar and time expenditures are related to changes in environmental quality. For example, in recent Mathtech work (Manuel et al., 1983) an attempt was made to estimate air pollution control soiling benefits to households by building a model that analyzed household expenditure decisions by major expense category. By assuming separability across categories, they could focus upon the cleaning related expenditures and how they varied as pollution levels varied. The approach has also been used to value human health impacts by relating expenditures by health care category to air pollutants (see Chestnut and Violette, 1985, for a review).

The advantage of this approach is that it can be well grounded in economic theory and, if sufficient data exist, it can be quickly applied. The principal limitation is that the required data do not exist for many types of impacts of interest.

A.7 CONTINGENT VALUATION APPROACHES

A.7.1 Contingent Bidding Method

In bidding methods surveys, respondents are given information concerning alternative levels of air quality, or of a good or service that embodies, or is affected by, air quality. They are also given hypothetical markets that describe how payments are made or received by the respondent that relate to the good or resource being valued. Next they are asked to bid their maximum WTP or minimum willingness to accept compensation to prevent or incur the change from one level of air quality (or of the good or service affected by air quality) to another (Davis, 1963; Randall et al., 1974; Cummings et al., 1985; and others).

The principal strength of this approach is that it can be used to address almost any air quality related issue, regardless of whether it has yet occurred or whether the respondent was previously aware of the issue. It can also be designed to estimate benefits separately for different effects as well as user and non-user benefits of resource protection.

Bidding methods have several significant design considerations. Important among these is the design of questions that minimize perception errors and biased responses. Bidding methods ask respondents to reveal what their behaviour would be in a hypothetical situation, not often faced in a market place. Only estimates of the "true" values and predictions of what their behaviour would be in the hypothetical situation, or market, as they perceive it, can be reported. The reported values may well reflect the respondents' "true" values estimated with uncertainty and subject to the influences inherent in the design of the survey instrument.

Survey instrument influences may decrease the accuracy of responses and may yield biases in the valuation process. The evidence to date suggests that the inaccuracies may or may not be substantial depending upon specifics of the application (Rowe and Chestnut, 1983; Cummings et al., 1985).

A.7.2 Contingent Ranking

The contingent ranking approach, as applied by Rae (1982) and by Desvousges et al. (1983), attempts to determine how respondents value changes in environmental conditions by asking them to rank alternative scenarios. The alternatives present different combinations of environmental attributes, perhaps at a site or embodied within another good or service, and market costs, such as utility bills, taxes, etc. By determining how changes in the environmental resource affect the probability that an alternative will be chosen, theoretically correct measures of benefits can be obtained. This is done by holding all variables constant and determining how prices must change to offset the change in environmental quality such that the probability of selecting an alternative from all competing alternatives remains constant.

A principal advantage of this approach is that respondents are not required to directly assign dollar values to the environmental resource, but reveal the value indirectly through rankings.

A.8 OTHER IMPLICIT VALUATION TECHNIQUES

Other techniques are available that, although often not directly related to economic welfare theory, may assist in quantifying resource protection benefits. One such approach is voting (described in more detail in Freeman 1979), where implicit

relative values are assumed to be revealed through voting results. For example, if a piece of legislation is passed to preserve a natural resource that will cost society \$x, in terms of pollution control costs or lost resource development income, then one might infer that the value of protecting the resource was revealed to exceed \$x. Similarly, legislation that merely delays impacts until more thorough evaluations are performed might reflect option prices related to resource protection. There are many such ways that voting behaviour can be analyzed. Similarly, market and non-market behaviour could be examined, including bequests, charitable donations, etc.

A.9 SELECTION OF STUDIES

The selection of studies used to value effects was generally based upon three criteria: applicability, theoretical validity, and presumed accuracy. The evaluation of available studies using these criteria was, however, based upon professional judgement rather than a standardized procedure.

Applicability refers to whether the study addresses impact category values of interest, such as recreational impacts from reduced visual aesthetics. Of those studies that were felt to be applicable, studies conducted in North America were ranked as most useful, conditional upon the remaining criteria.

Theoretical validity refers to whether a study uses methods and assumptions consistent with physical laws and economic theory, and produces an economic measure of value that is consistent with the conceptually correct concepts and measures. If these conditions are not met, interpretation of the physical and economic estimates is often difficult. In many cases, the estimates produced by methods that are not theoretically valid

may be biased or have no relationship to underlying economic concepts. Theoretically valid studies are obviously preferred, although other approaches such as the damage function approach may still be useful as the direction of bias is generally known.

Accuracy refers to the ability of the methods employed in a study to produce accurate estimates of the "true" value of interest. The true value, however, is generally unknown. For this reason, judgments of accuracy are often based upon the consistency of a study's procedures and results with other similar efforts, and known strengths and weaknesses of the methods used.

A.10 THE VALUE OF REDUCING RISKS OF DEATH¹

Many regulations impose costs on society in exchange for reducing the risks of death. Reducing risks by a small amount for each of many people can add up to many lives saved. Several methods have been proposed for generating estimates of the value of reducing risks of death that can be contrasted with the costs of obtaining such reductions. The current consensus in the economics profession is that the appropriate way to measure this value is to determine what people are willing to pay.

A willingness to pay (WTP) estimate values the change in well-being that would result from changing the risk of death. It is measured by how much of other goods and services a person is willing to give up to get that reduction in the risk of death.

¹ This section is based on a paper forthcoming in the Journal of Policy Analysis and Management entitled "The Value of Reducing Risks of Death: A Note on New Evidence", by Ann Fisher, Lauraine G. Chestnut and Daniel M. Violette.

Summing this measure across all people can provide an estimated value of a statistical life. Rather than the value for any particular individual's life, the value of a statistical life represents what the whole group is willing to pay for reducing each member's risk by a small amount. For example, if each of 100,000 persons is willing to pay \$20 for a reduction in risk from 3 deaths per 100,000 people to 1 death per 100,000 people, the total WTP is \$2 million and the value per statistical life is \$1 million (with 2 lives saved).

Innovative methods must be used to determine what people are willing to pay for reductions in risks of death, because risk is not directly traded in markets. WTP estimation studies can be grouped into three categories:

- . wage-risk studies;
- . contingent market studies; and
- . consumer market studies.

Exhibit A.7 summarizes results from selected studies in each category. All results are presented in 1986 U.S. dollars. In addition to the WTP estimates, the exhibit shows the mean level of the risk considered in each study; the level of risk faced by the individual is likely to influence his willingness to pay to reduce risk. The sources give additional information about each study cited in the exhibit.

A.10.1 Wage-Risk Studies

Wage-risk studies estimate the wage premium associated with greater risks of death on-the-job. Suppose jobs A and B are identical except that workers in job A have higher annual fatal injury risks such that, on average, there is one more job-related death per year for every 10,000 workers in job A than in job B, and workers in job A earn \$500 more per year than those in job B.

EXHIBIT A.7: ESTIMATES OF THE MARGINAL WILLINGNESS TO PAY FOR REDUCTIONS IN RISKS

Study	Mean Risk Level for The Sample ^a	(millions of U.S. 1986 dollars)	
		Range of Estimates	Judgemental Best Estimate
<u>EARLY LOW-RANGE WAGE-RISK ESTIMATES</u>			
1. Thaler and Rosen (1975) ^b	11.0	0.44-0.84	0.64
2. Arnould and Nichols (1983) ^b	11.0	0.72	0.72
3. Dillingham (1979)	1.7	0.38-1.2	0.45
<u>EARLY HIGH-RANGE WAGE-RISK ESTIMATES</u>			
(all based on BLS industry accident rates)			
4. R. Smith (1974)	1.0-1.5	8.5-14.9	8.5
5. R. Smith (1976)	1.0 & 1.5	3.6-3.9	3.7
6. Viscusi (1978)	1.2	4.1-5.2	4.3
7. Olson (1981)	1.0	8.0	8.0
8. Viscusi (1981)			
a. w/o risk interaction terms	1.04	5.4-7.0	7.0
b. with risk interaction terms	1.04	4.7-13.4	
9. V.K. Smith (1976) ^c	3.0	1.9-5.8	3.9
<u>NEW WAGE-RISK STUDIES</u>			
10. Dillingham (1985)	1.4-8.3	2.1-5.8	2.5
11. Marin & Psacharopoulos (1982) ^d			
a. Manual workers	2.0	2.7-3.1	2.9
b. nonmanual workers	2.0	9.0	
12. Low and McPheters (1983) ^e	3.6	0.9	0.9
13. Leigh and Folsom (1984)	1.3-1.4	4.3-10.2	6.8
14. Gegax et al. (1985)			
a. all union workers	8.2	1.9	
b. union blue-collar workers	10.1	1.6	1.6
15. Moore and Viscusi (1988)	0.79 ^f	5.2-6.6	5.4
	0.52 ^g	1.9-2.1	
<u>NEW CONTINGENT VALUATION STUDIES</u>			
16. Jones-Lee et al. (1985)	0.8-1.0	1.6-4.4	3.0
17. Gegax et al. (1985)	4.2-10.0	2.4-3.3	2.8
<u>CONSUMER MARKET STUDIES</u>			
18. Ghosh, Lees, and Seal (1975)	not reported	0.56	.56
19. Blomquist (1979)	3.0	0.38-1.4	.61
20. Dardis (1980)	0.9	0.36-0.56	.46
21. Ippolito and Ippolito (1984)	Varied	0.24-1.26	.52

Notes:

- a. Approximate annual deaths per 10,000 people.
- b. Based on actuarial risk data.
- c. Assuming 0.4 percent of all injuries are fatal, as reported by Viscusi (1978) for the BLS injury statistics, and that the risk premium for fatal injuries is 33-100 percent of the premium for all risks.
- d. Their age-adjusted normalized risk variable is not directly comparable to the risk levels used in other studies. However, the average risk of death for the entire sample was 2 in 10,000.
- e. Four years of risk data were used. the overall mean was not reported, but the mean for one year was 3.6 deaths out of 10,000 workers across all cities in the sample.
- f. This row is based on NIOSH National Traumatic Occupational Fatality data by one-digit SIC code for each state.
- g. This row is based on BLS accident rates by two-digit SIC code.

EXHIBIT A-7: ESTIMATES OF THE MARGINAL WILLINGNESS TO PAY FOR REDUCTIONS IN RISKS (Continued)

SOURCES:

Richard J. Arnould and Len M. Nichols, "Wage-Risk Premiums and Worker's Compensation: A Refinement of Estimates of Compensating Wage Differential," Journal of Political Economy. (91) (April 1983): 332-340.

Glenn Blomquist, "Value of Life Savings: Implications of Consumption Activity," Journal of Political Economy. (87) (June 1979): 540-558.

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Douglas Gegax, Shelby Gerking, and William Schulze, "Perceived Risk and the Marginal Value of Safety," working paper prepared for the U.S. Environmental Protection Agency, (August 1985).

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P.M. Ippolito and R.A. Ippolito, "Measuring the Value of Life Saving from Consumer Reactions to New Information," Journal of Public Economics. (25) (1984): 53-81.

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J. Paul Leigh and Roger N. Folsom, "Estimates of the Value of Accident Avoidance at the Job Depend on the Concavity of the Equalizing Differences Curve," Quarterly Review of Economics and Business. (24) (Spring 1984): 56-66.

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Alan Marin and George Psacharopoulos, "The Reward for Risk in the Labor Market: Evidence from the United Kingdom and a Reconciliation with Other Studies," Journal of Potential Economy. (90)4 (1982): 827-853.

Michael J. Moore and W. Kip Viscusi, "Doubling the Estimated Value of Life: Results Using New Occupational Fatality Data," Journal of Policy Analysis and Management. (7)3 (Spring 1988): 476-490.

Craig A. Olson, "An Analysis of Wage Differentials Received by Workers on Dangerous Jobs," Journal of Human Resources. (16) (1981): 167-185.

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V. Kerry Smith, "The Role of Site and Job Characteristics in Hedonic Wage Models," Journal of Urban Economics. (13) (1983): 296-321.

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W. Kip Viscusi, "Occupational Safety and Health Regulation: Its Impact and Policy Alternatives," in Research in Public Policy Analysis and Management Vol. 2, J. Creecine, Ed., (Greenwich, CT: JAI Press, 1981): 281-299.

The implied value of a statistical life is then \$5 million for workers in job B who are each willing to forego \$500 per year for a 1 in 10,000 lower annual risk. Jobs typically differ by other factors as well, so regression analysis is used with wage-risk data to account for these other influences.

The wage-risk approach relies on several assumptions. First, it assumes workers are aware of differing risks across jobs. If workers do not perceive risk accurately, this will reduce the significance of the estimated coefficient on the risk variable. Second, it assumes workers are able to move freely between jobs. Questions still remain about how institutional constraints in the labour market affect the estimates that are obtained under the assumptions of effective competition and full information. Some, but not all, of the wage-risk studies account for job characteristics other than risk also could affect wage differentials.

The wage-risk studies have several potential limitations. Most do not treat compensation for nonfatal risks as part of the wage-risk premium. This is probably reasonable because worker's compensation insurance often covers a portion of the costs of nonfatal injuries. Most wage-risk studies rely on wages rather than measures of total compensation. Finally, questions remain about the statistical estimation procedures used in these studies.

Early Wage-Risk Results

Data on job risks are scarce, and a major criticism of the early studies has been that most of them used one of only two sets of data on job risks. The estimated values per statistical life are either low or high, depending on which of the two risk data sets was used.

The first two studies in Exhibit A.7 are based on actuarial risk estimates compiled from insurance data. All of the early wage-risk studies that used the actuarial type of risk measure obtained estimates in the low range. All of the early wage-risk studies that obtained results in the high range used the Bureau of Labour Statistics (BLS) data on work-related fatalities by industry group.

New Wage-Risk Results

Recent wage-risk studies have used new data sets and addressed some of the questions raised about earlier wage-risk analyses. Their results tend to confirm the empirical estimates in the upper range.

A.10.2 Contingent Valuation Studies

The contingent valuation approach poses a hypothetical market situation to survey respondents who then are asked about their willingness to pay for alternative levels of safety. Its principal disadvantage is that it is based on what people say, rather than on what they do.

Early contingent valuation studies for valuing changes in risks had several limitations and have not been included in Exhibit A.7. There have been substantial advances in the use of the contingent valuation approach over the past few years, however. Two new contingent valuation studies that used state-of-the-art methods to value change in the risk of fatality are summarized in the exhibit.

The consistency between new contingent valuation estimates and those obtained from the wage-risk analyses partially refutes the criticism that the contingent valuation results are not credible because they are based on what people say rather than what they do.

A.10.3 Consumer Market Studies

Consumer market studies examine the observable tradeoffs people make between risks and benefits in their consumption decisions. These studies analyze actual behaviour and because of this are similar to wage-risk studies. The consumer market applications conducted so far, however, have some important limitations regarding their ability to provide credible estimates of the value of a statistical life.

The results for consumer market studies of smoke detectors, use of automobile seat belts, speeding behaviour by drivers, and reactions to information about risks from smoking are presented in Exhibit A.7. The first three studies were designed to test whether the market actually responds to differences in safety rather than to estimate the value of a statistical life, but such estimates can be inferred from their results. The results are similar in magnitude to the low estimates of the early wage-risk studies.

The fourth study estimated the value of reducing the risk of death based on the reduction in smoking that has occurred in response to new information about smoking risks. The unique nature of the risk makes it difficult to interpret this study, however. Smoking is an activity that is habit forming. Reducing its risks involves significant physical and psychological costs for many individuals. Therefore, estimates based on reduced smoking would understate the value of reducing risks of a more generic nature.

A.10.4 Conclusions

The most defensible empirical results indicate a range for the value-per-statistical-life estimates of \$1.6 million to \$8.5 million (in 1986 U.S. dollars). The figure we selected for the bottom of this range was chosen for three reasons. First, strong evidence shows that the lower value-of-life estimates obtained from early wage-risk studies result from biases in the measured risk variable and should not be included in the range of empirical estimates. Second, the study of police officers (which is the only new wage-risk study with results below \$1.6 million) probably traces a labour supply curve rather than showing wage differentials for a risky occupation. Third, the results of consumer market studies conducted to date, which fall below the \$1.6 million estimate, reflect potentially significant downward biases due to the assumptions that underlie each of the analyses.

Less can be said about the firmness of \$8.5 million as an appropriate upper end on the range. All of the "best" estimates shown in Exhibit A.7 above about \$5.5 million are from wage-risk studies that have used the BLS risk data. The wage-risk studies that yielded estimates in the upper part of the range may not have controlled adequately for job characteristics other than risk. In addition, values derived from the wage-risk studies may overstate the value of reducing risks of death for elderly people whose life expectancies are shorter than typical employed individuals.

There are other potential benchmarks to check the reasonableness of the value-per-statistical-life range. One benchmark -- human capital estimates -- looks at what the "saved" individual contributes to society through his or her future earnings. A major drawback of this approach is that it assigns lower values to the lives of women and minorities, and no value to the lives

of retired people. Its crucial shortcoming is that it does not reflect the change in well-being to each person (among the many people) who experiences the small reduction in risk of fatality.

A second benchmark could be what actually is spent to rescue people from life-threatening situations, such as coal miners trapped by a cave-in. Most regulations that affect risk reduce the risk by a small amount for each of many people. There is no way to identify which particular individuals will be served by the regulation. In contrast, the coal mine operators know who is trapped by the cave-in. Empathy for these identifiable individuals typically leads to extraordinary rescue efforts. Casual observation suggests, that these outpourings of effort and resources do not transfer to saving unidentifiable lives.

Purchases of life insurance have been suggested as a third potential benchmark. One study noted that premiums are about 1.6 times the value of claims for a representative term life insurance policy. Life insurance, however, is purchased primarily to protect the standard of living of the beneficiaries; the premiums do not reduce the policy holder's risk of death. Such purchases cannot, therefore, be expected to reflected the value of reducing fatality risks.

The uncertainties in the results of the studies presented indicate that the \$1.6 million to \$8.5 million range should be viewed as an interim range. We will use as well a central value of \$5.0 million.

A.11 ECONOMIC VALUE OF REDUCED DAMAGES

Our review of the literature reveals wide ranges for the economic values estimated for specific categories of reduced damages. These are shown in Exhibit A.8.

The range for restricted activity days is similarly broad with a central value of \$60 and upper and lower bounds of \$70 and \$15 respectively.

The benefits for hospital admissions for respiratory disease, materials damage and visibility are estimated directly from the exposure response functions. These are presented in Appendix H.

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EXHIBIT A.8: VALUES USED FOR CALCULATING ECONOMIC BENEFITS OF
AVOIDING PARTICULAR TYPES OF DAMAGE

Benefit Category	Value	Value
	1986 U.S. \$	1986 C \$
Reduced Risk of Death		
central	5,000,000	6,950,000
lower	1,600,000	2,200,000
Emergency Room Visits		
central	175	250
Restricted Activity Days		
upper	52	70
central	44	60
lower	12	15
Hospital Days for Respiratory Conditions	1,050	1,450
Hospital Admissions for Respiratory Disease	6,500	8,580
Materials Damage	*	*
Visibility	*	*

Notes: * represents damages that are not quantified in damage functions on a per event basis.

Conversion rate: 1986 U.S. to 1986 Canadian dollars
= 1.39.

EXHIBIT A.8: VALUES USED FOR CALCULATING ECONOMIC BENEFITS OF
AVOIDING PARTICULAR TYPES OF DAMAGE (Continued)

Sources:

- . Reduced risk of death: see Exhibit A.7.
- . Emergency room visits:

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APPENDIX B

Economic Sectors

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B.1 INTRODUCTION

This appendix gives an overview of the economic sectors considered in this study. These sectors are:

- . Open Pit Mining;
- . Food Industries;
- . Pulp and Paper and Allied Products;
- . Iron and Steel;
- . Foundries;
- . Non-Ferrous Smelters and Refineries;
- . Motor Vehicle Industries;
- . Inorganic Chemical Manufacturing;
- . Petroleum Refineries;
- . Asphalt Paving;
- . Petrochemical Products;
- . Power Generation;
- . Waste Disposal;
- . Dry Cleaners; and
- . Restaurants.

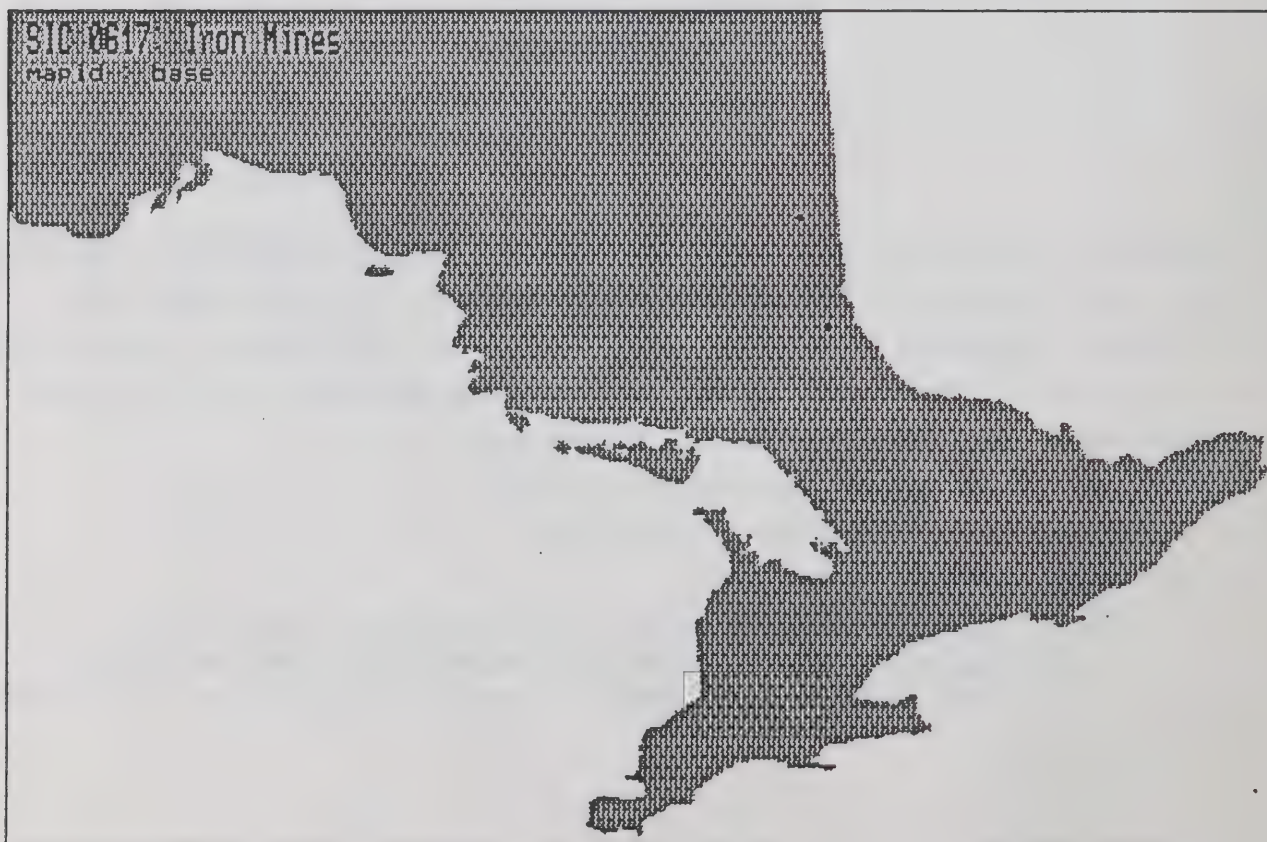
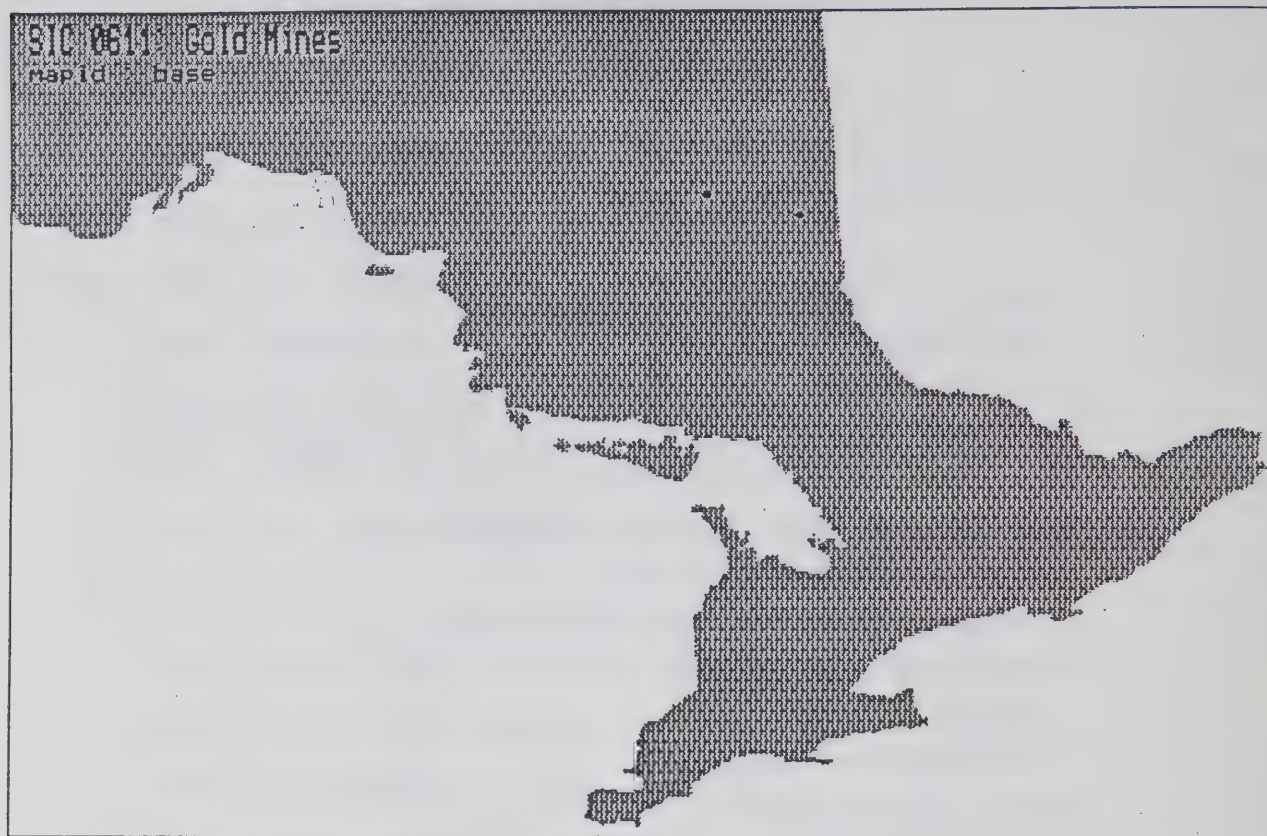
B.2 OPEN PIT MINING

Most of the metal ore mines in Ontario are underground. A list of open pit mines and their locations was obtained from the Ontario Ministry of Northern Development and Mines. There are a total of six open pit mines, all in the northern part of the province (see Exhibit B.1). These are:

- . 4 - Gold Mines (SIC-0611)¹; and
- . 2 - Iron Ore Mines (SIC-0617).

¹. The SIC codes are the 1980 Standard Industrial Classification codes as defined Statistics Canada. They differ from the U.S. and 1970 Canadian SIC codes.

EXHIBIT B.1: OPEN PIT MINING



The total employment of the four open pit gold mines and the two open pit iron ore pit mines is about 1910 workers. Open pit mines are sources of particulate emissions. Emissions at each location are estimated by apportioning the total emissions for the sector across the mines identified, on the basis of the number of employees at each mine site.

B.3 FOOD INDUSTRIES

The analysis covers four different food industries:

- . Meat and Meat Products (SIC-1011);
- . Poultry Products (SIC-1012);
- . Fish Products (SIC-1021); and
- . Other Dairy Products (SIC-1049).

The last industry (SIC-1049) is limited to the seven plants that dry whey. The plants that comprise these four industries are primarily situated in southwestern and southcentral Ontario (see Exhibits B.2 and B.3). The largest of these industries is meat and meat products. We list a total of 114 establishments employing 8,077 workers based on the MITT Made in Ontario data base and Statistics Canada industry information. Emissions by establishment are estimated from industry emissions using the ratio of plant employment to industry employment.

The poultry products industry in Ontario consists of 32 establishments. Our list was obtained by taking all establishments listed in Agriculture Canada's Meat Hygiene Manual, Part B, that only process poultry. Employment figures were not available. The estimated emissions for the industry were apportioned equally among the 32 establishments.

EXHIBIT B.2: FOOD INDUSTRY, MEAT AND POULTRY PRODUCTS

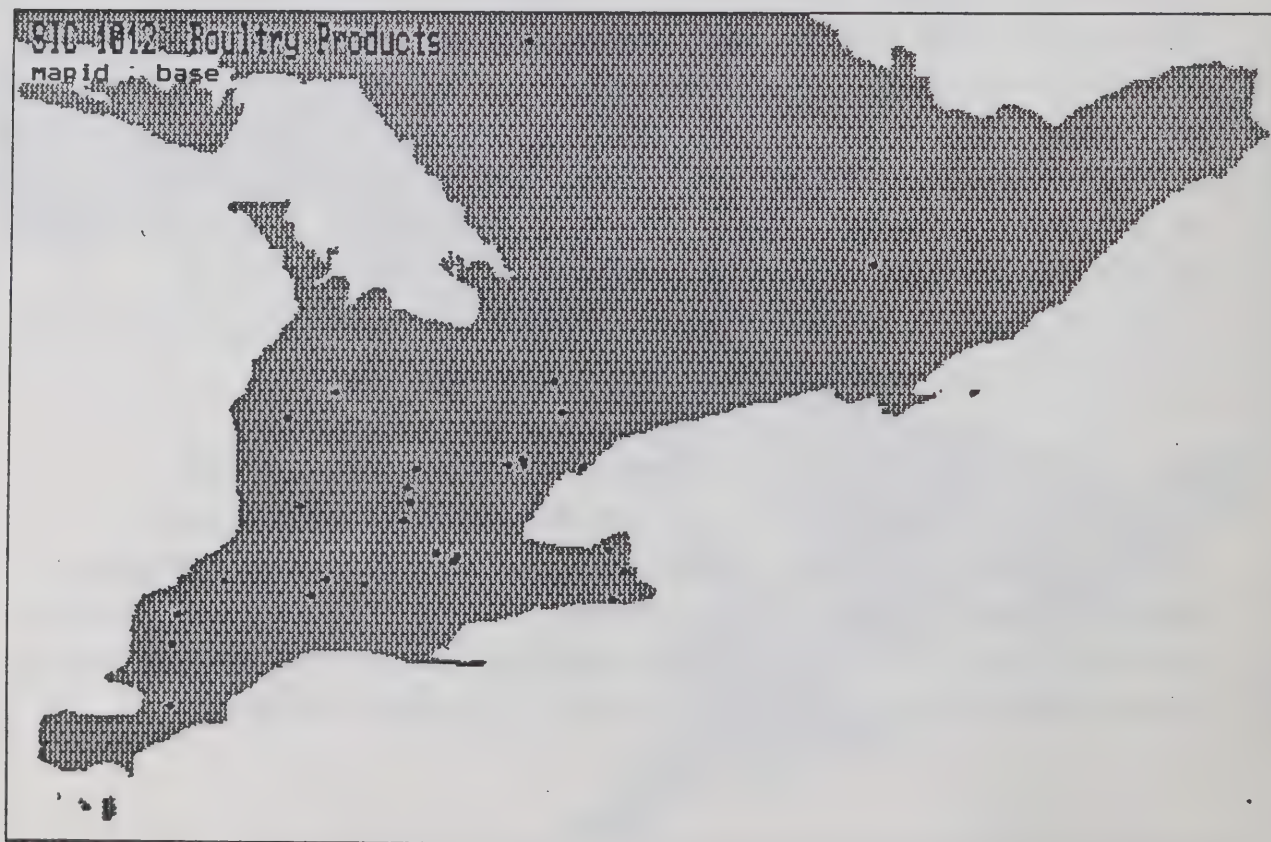
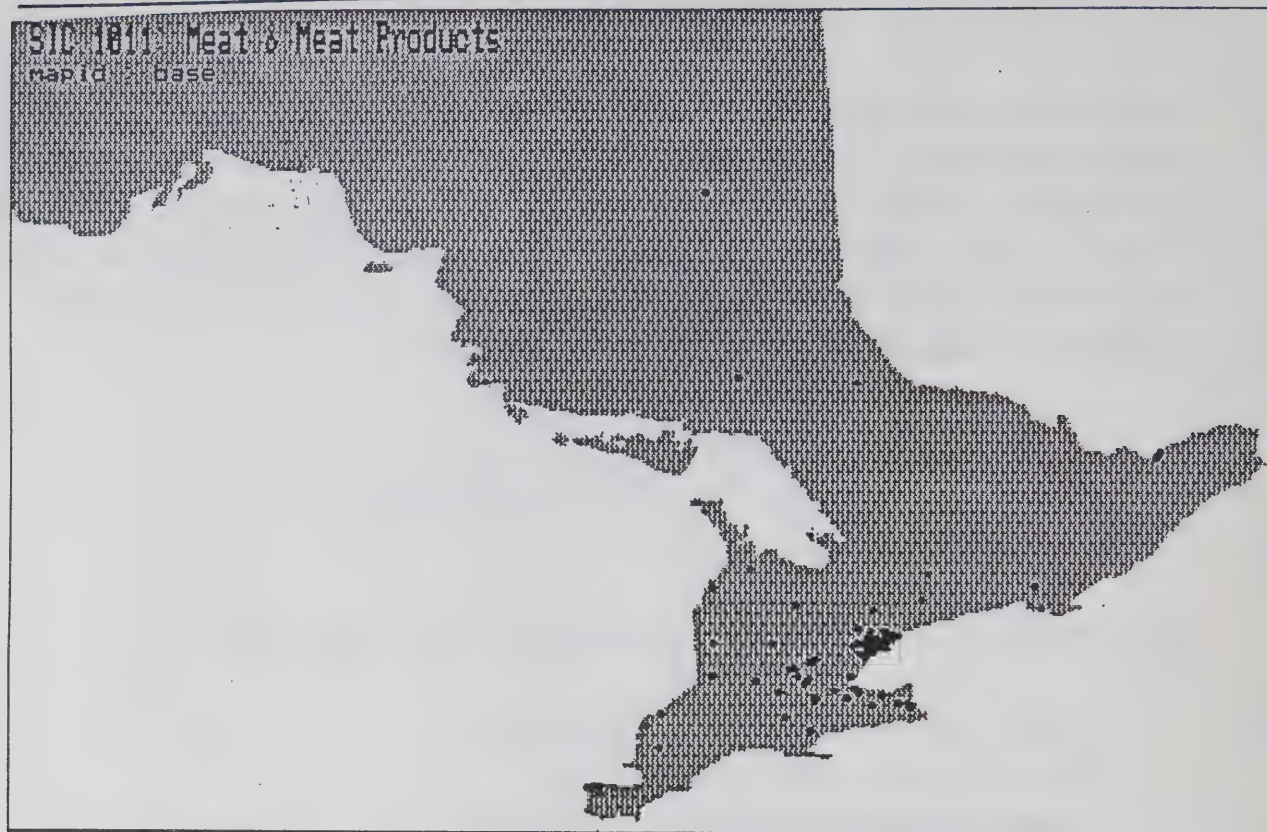
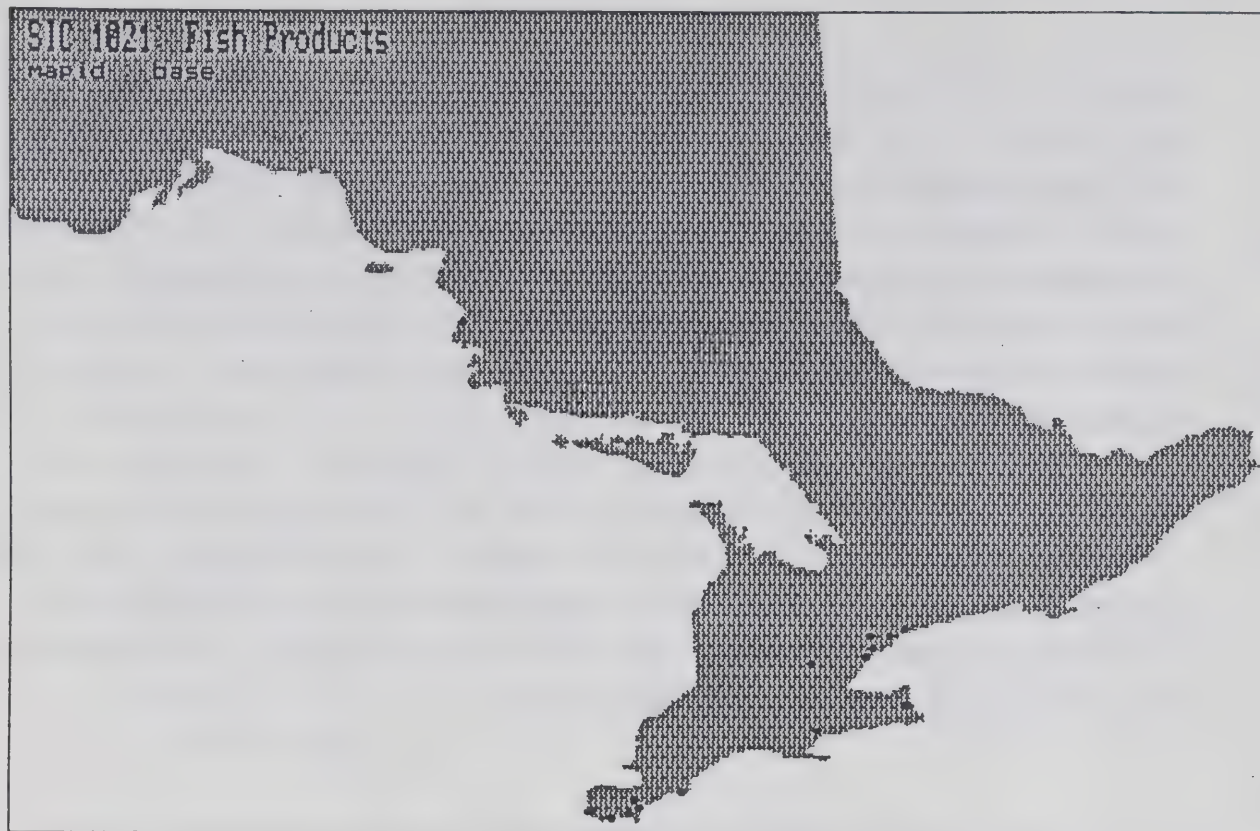


EXHIBIT B.3: FOOD INDUSTRY, FISH PRODUCTS AND WHEY DRYING



Ontario does not have a major fishing industry. This is reflected in the small number of plants. We identify 17 facilities with 551 employees. Emissions at individual establishments are estimated from total industry emissions using employment at the plant in relation to total employment. The major contaminants emitted by the meat, poultry and fishery products industries are volatile organic compounds (VOC's) and particulate matter (PM).

There are seven establishments with 551 workers that dry whey in Ontario. This list was compiled through consultation with the Canadian Dairy Council, Hickling Management Consultants and industry representatives. The major contaminants emitted during the drying of whey are particulates.

B.4 PULP AND PAPER AND ALLIED PRODUCTS

B.4.1 Pulp and Paper Industry

The pulp and paper industry has three components:

- . Pulp Industry (SIC-2711);
- . Newsprint Industry (SIC-2712); and
- . Paperboard Industry (SIC-2713).

Several newsprint and paperboard mills also have pulping facilities. The composition of the emissions is heavily influenced by the pulping process used. The principal pulping processes are:

- . kraft;
- . sulphite;

- . semi-chemical; and
- . groundwood.

The listings for all three industries were compiled from the Made in Ontario data base and Statistics Canada list of establishments. A few individual plants were shifted between the pulp industry and the newsprint industry to more accurately reflect the production of pulp. The pulp and newsprint mills are concentrated in northern Ontario while the paperboard mills are predominantly situated in southern Ontario (see Exhibits B.4 and B.5).

In total there are 36 pulp and paper establishments in Ontario. This total is made up of 9 pulp mills (7,207 employees), 11 newsprint mills (6,185 employees), and 16 paperboard mills (2,737 employees).

The major contaminants emitted by the pulp and paper industries are sulfur dioxide (SO₂), particulates (PM,) nitrogen oxides (NO_x), chlorine and chlorine dioxide. In each case industry emissions are divided among individual plants on the basis of employment.

B.4.2 Printing and Publishing Industries

The printing and publishing sector includes five industries:

- . Business Forms Printing (SIC-2811);
- . Other Commercial Printing (SIC-2819);
- . Plate Making, Typesetting and Bindery (SIC-2821);
- . Newspaper, Magazine and Periodical Publishing (SIC-2841);
and
- . Other Combined Publishing and Printing Industries
(SIC-2849).

EXHIBIT B.4: PULP AND PAPER AND ALLIED PRODUCTS INDUSTRY

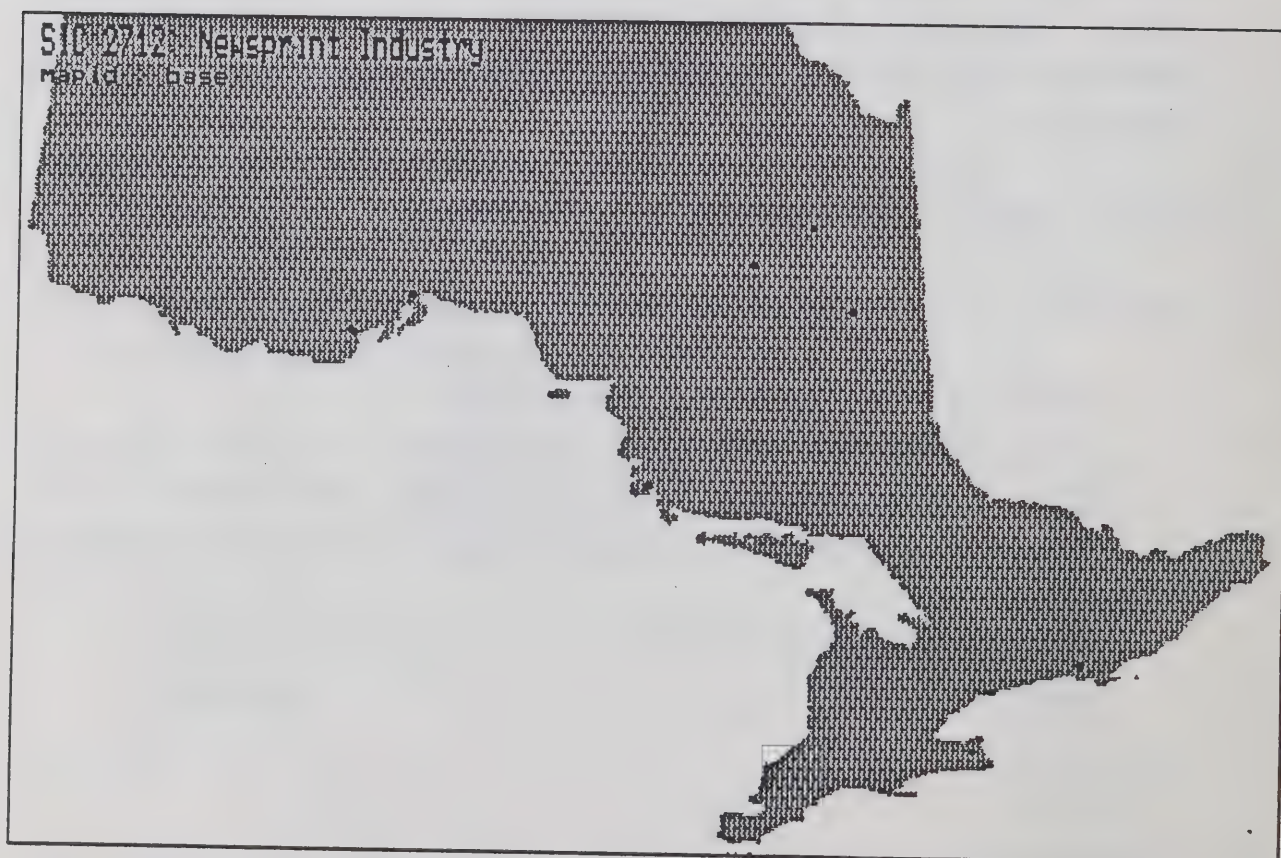
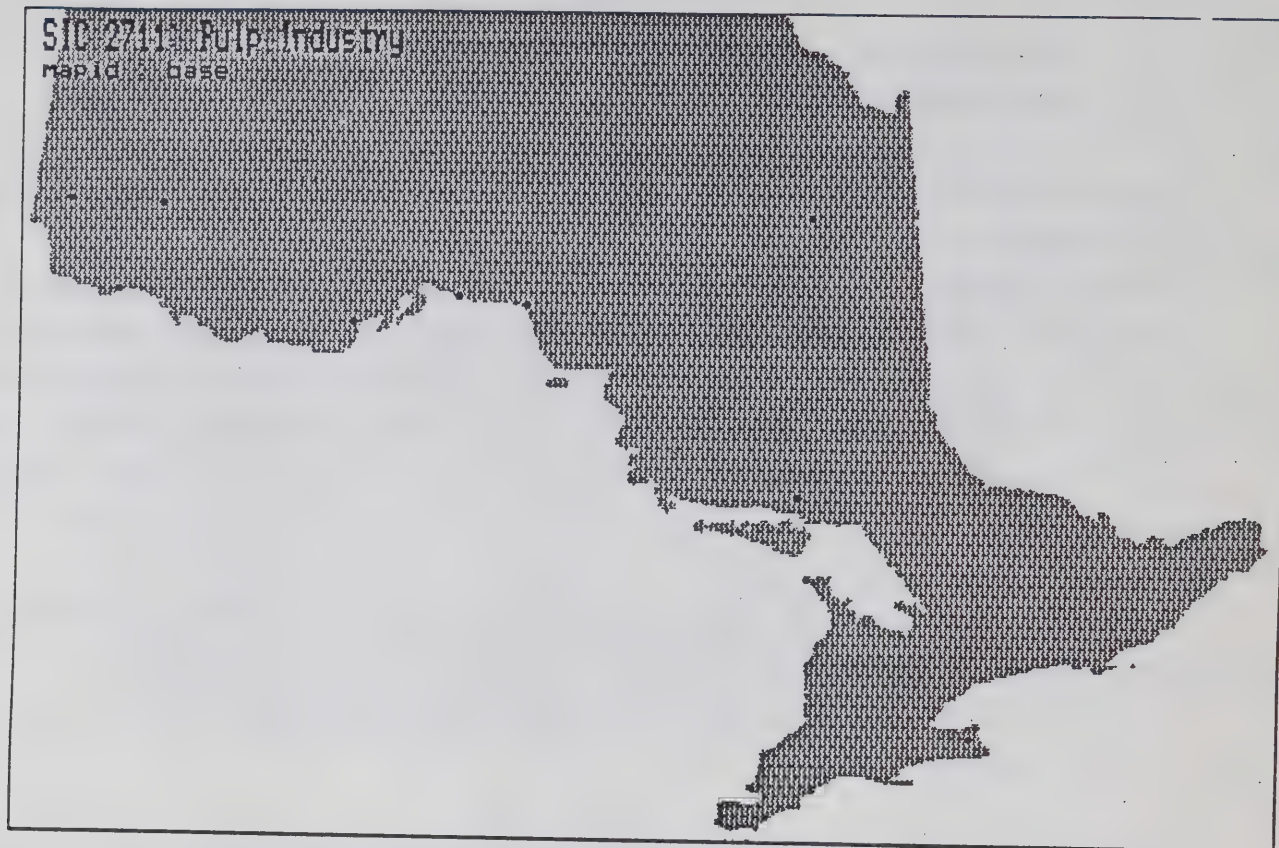
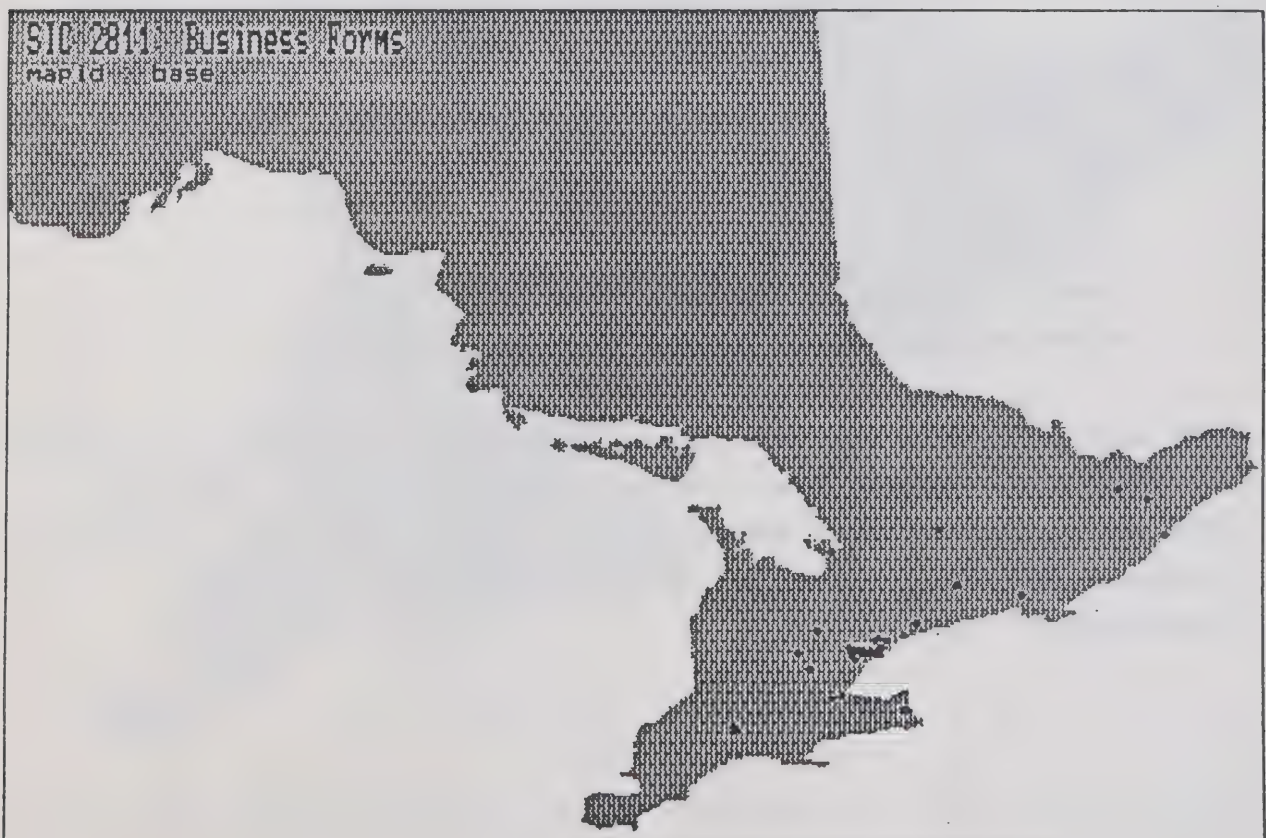
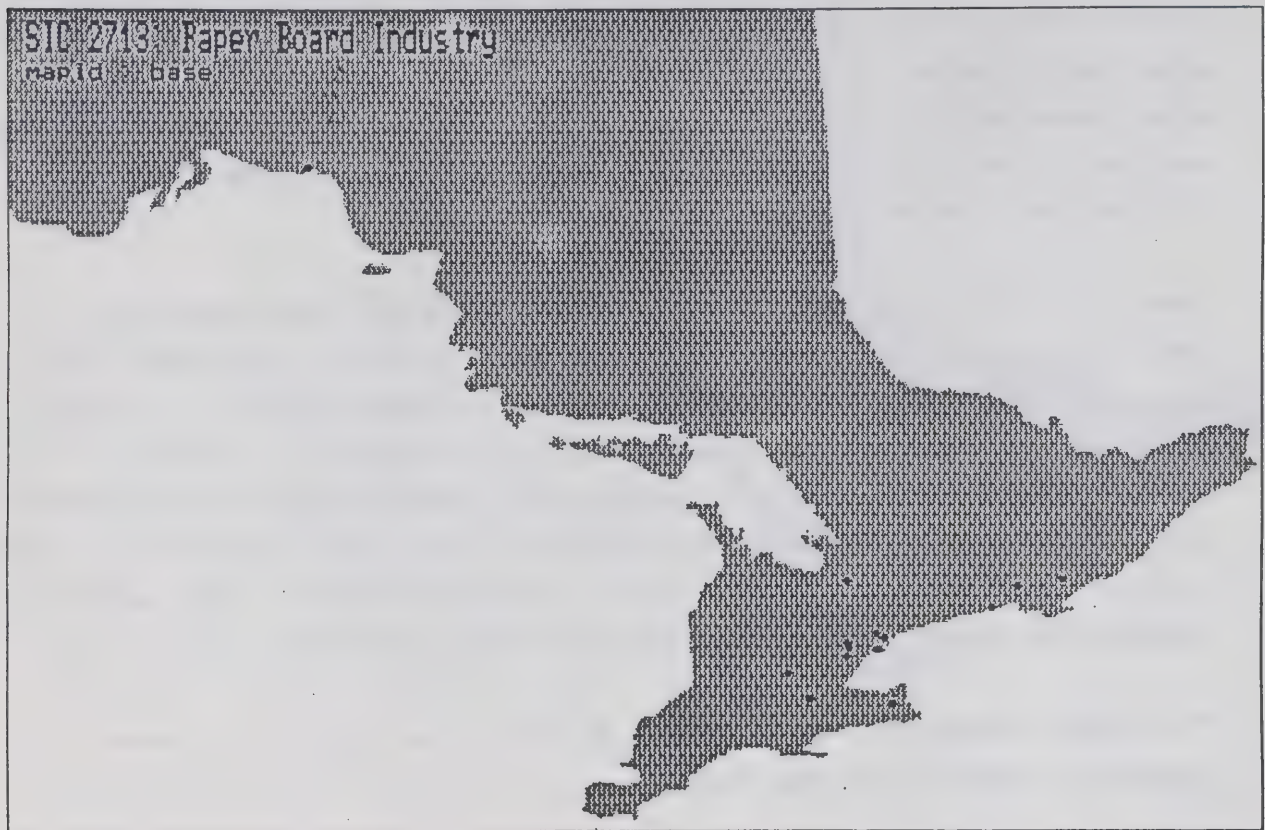


EXHIBIT B.5: PULP AND PAPER AND ALLIED PRODUCTS INDUSTRY



Together these five industries cover 621 establishments with a total of 46,695 employees. The printing and publishing industries are concentrated in Southern Ontario, particularly the Toronto area (see Exhibits B.5, B.6 and B.7). The list of establishments was compiled from the Made in Ontario data base and the Statistics Canada list of establishments.

Over half of the establishments (356) are in the commercial printing industry. They employ 23,601 workers. The next largest segment is the plate making, typesetting and bindery industry with 129 establishments employing 6,133 workers. We list 81 newspaper, magazine and periodical publishing establishments with 12,205 employees. The business forms printing has 44 plants (4,228 employees) and there are 11 establishments (528 employees) engaged in other combined publishing and printing activities.

The major contaminants emitted by all five industries are volatile organic compounds.

B.5 IRON AND STEEL INDUSTRIES

There are three industries in this sector:

- . Ferro-Alloys (SIC-2911);
- . Steel Foundries (SIC-2912); and
- . Other Primary Steel Industries (SIC-2919).

The list of plants for all three industries was compiled from information obtained from the MITT Made in Ontario and the Statistics Canada data bases. The establishments are located in southern Ontario, primarily southwestern Ontario (see Exhibits B.8 and B.9).

EXHIBIT B.6: PULP AND PAPER AND ALLIED PRODUCTS INDUSTRY



EXHIBIT B.7: PULP AND PAPER AND ALLIED PRODUCTS INDUSTRY

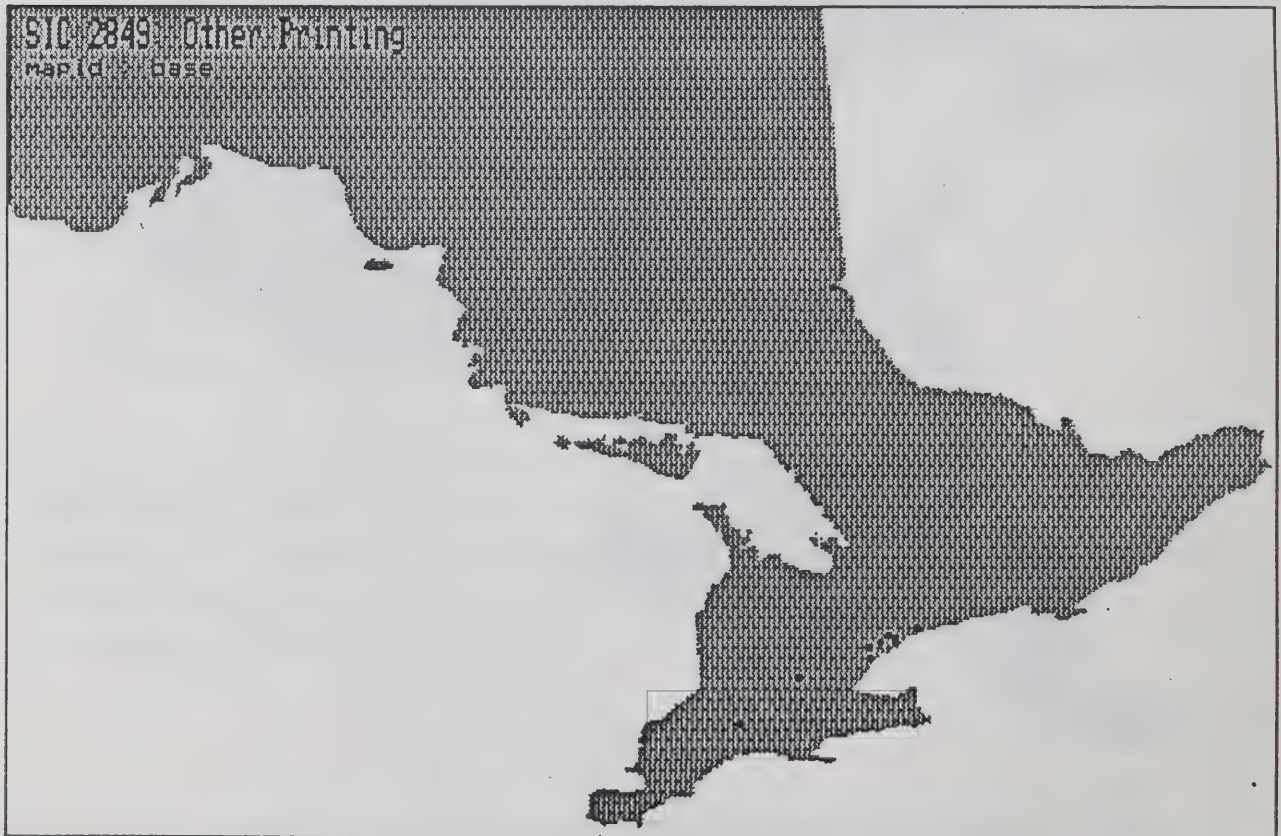
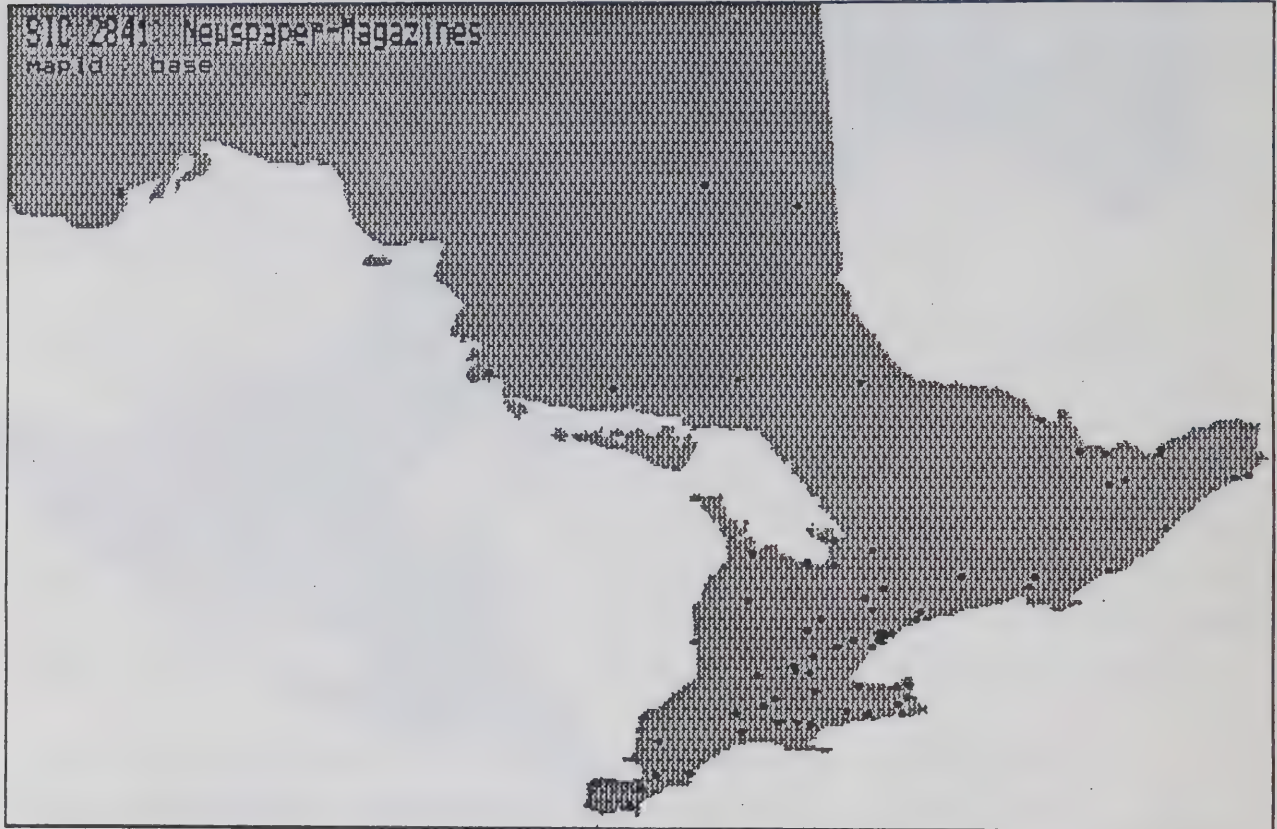
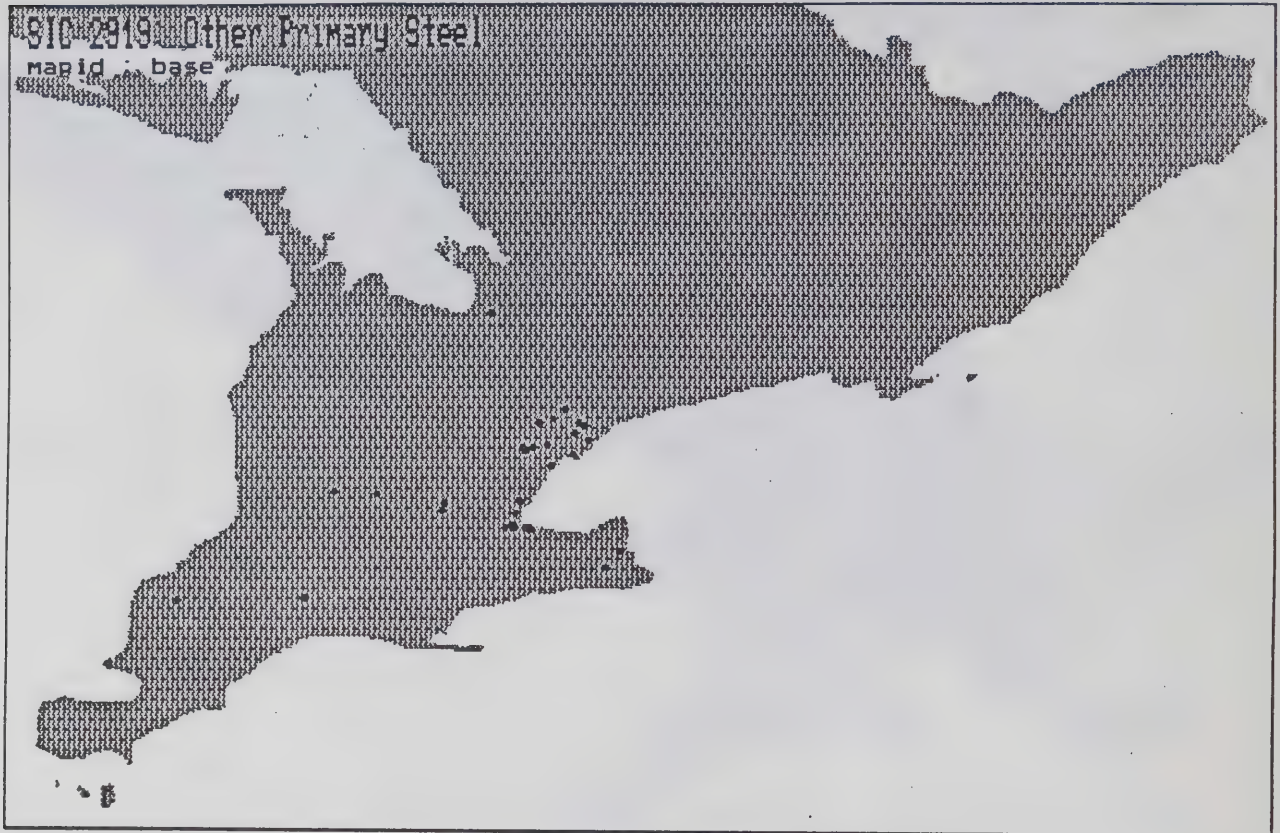


EXHIBIT B.8: IRON AND STEEL INDUSTRIES



EXHIBIT B.9: IRON AND STEEL INDUSTRIES



There is only one ferro-alloy plant in Ontario. It employs 16 people and emits particulate matter and volatile organic compounds.

The list of steel foundries was adjusted to include all major primary steel producers and foundries. Thus, we identified 22 establishments employing 39,9009 workers. The major contaminants emitted are particulate matter. A total of 35 plants (2,203 employees) were identified in other primary steel industries. The major contaminant emissions are volatile organic compounds, SO₂, and particulate matter.

For the steel foundries and other primary steel industries, emissions by each facility are estimated from industry emissions in the ratio of facility employment to industry employment

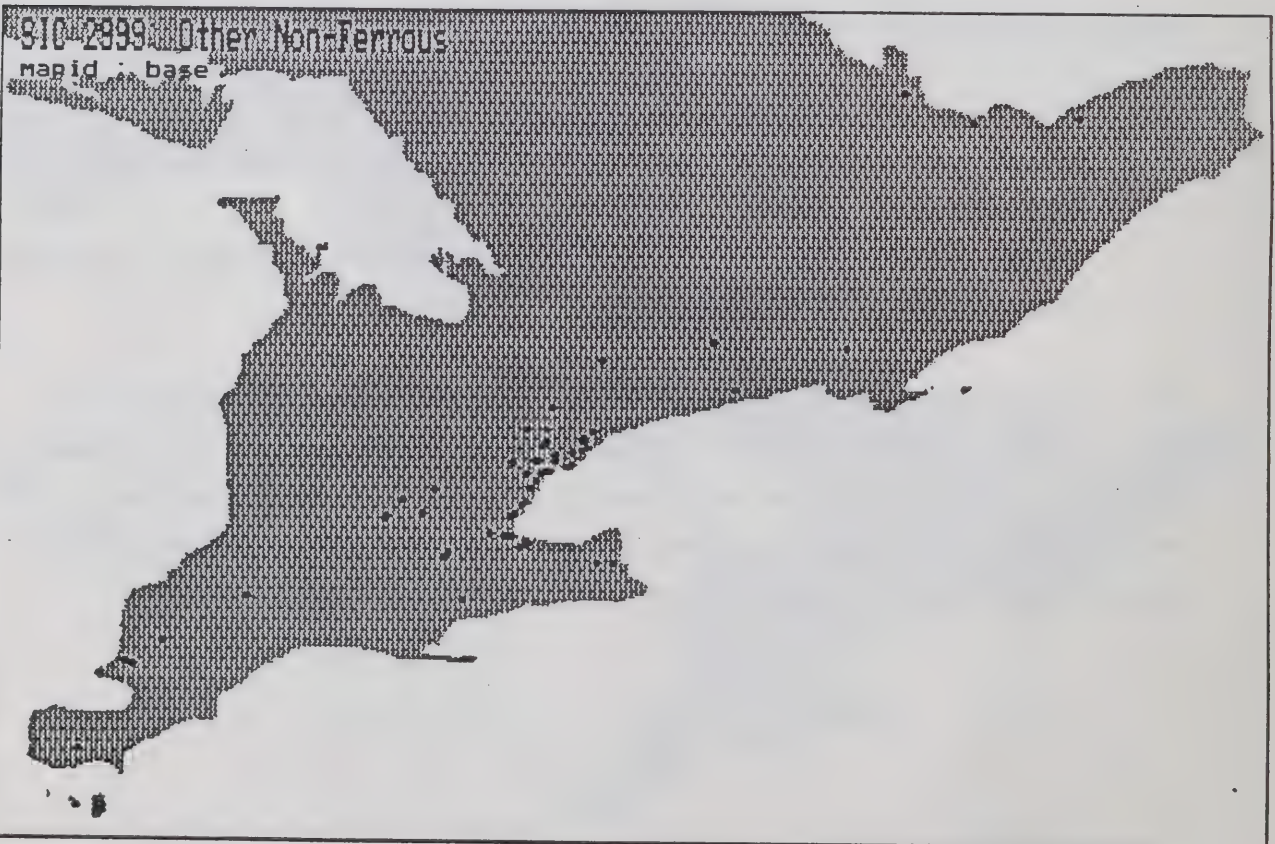
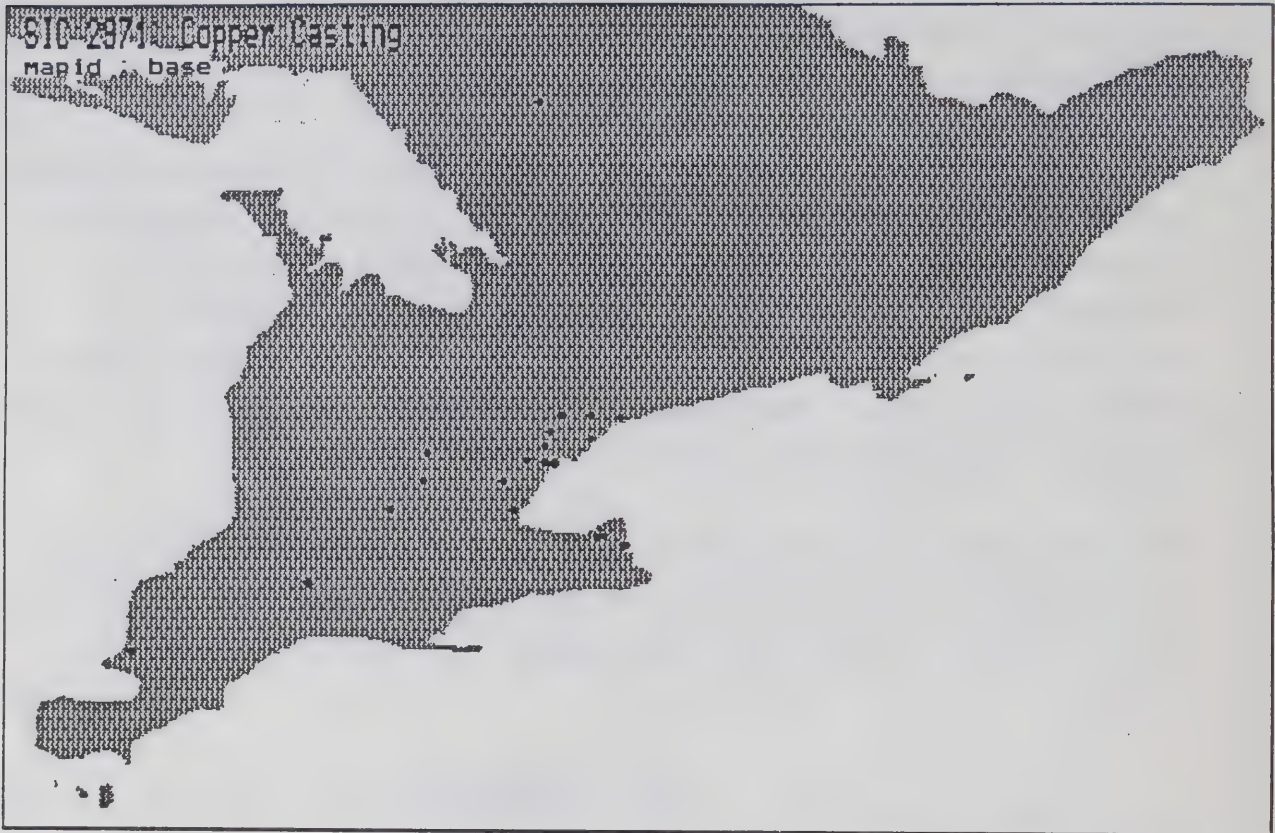
B.6 FOUNDRIES

Three industries were included in this section:

- . Iron Foundries (SIC-2941);
- . Copper and Copper Alloy Rolling, Casting and Extruding (SIC-2971); and
- . Other Rolled, Cast and Extruded Non-ferrous Metal Products Industries (SIC-2999).

All three industry listings were compiled from information obtained from the MITT Made in Ontario and Statistics Canada lists. Individual establishment emissions are based on plant employment. The establishments are located in southern Ontario, primarily the southwest (see Exhibits B.9, and B.10).

EXHIBIT B.10: NON-FERROUS FOUNDRIES



There are 61 iron foundries (7,110 workers) and 23 plants (2,149 workers) in the copper and copper alloy rolling, casting and extruding industry. Both of these industries emit particulate matter.

The 63 establishments involved in other rolled, cast and extruded non-ferrous metal products industries employ 5,623 workers and emit particulate matter and volatile organic compounds.

B.7 NON-FERROUS METAL SMELTING AND REFINING

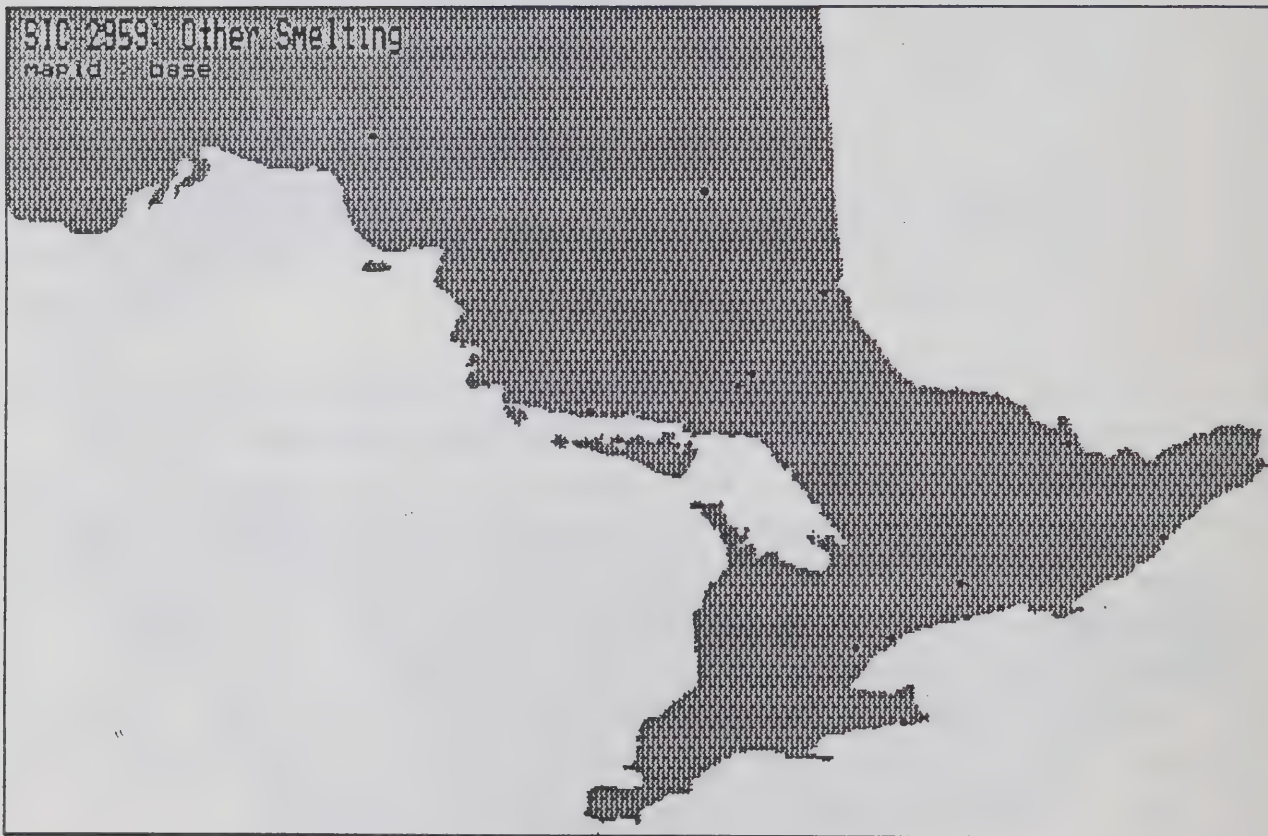
This sector is defined as:

- . Other Primary Smelting and Refining of Non-Ferrous Metal (SIC-2959).

Primary smelting and refining establishments were identified from the Made in Ontario data base and Statistics Canada information. Exhibit B.11 shows that this industry is located throughout the province.

We identified 16 other primary smelting and refining of non-ferrous metal establishments employing 7,761 workers. The major contaminants emitted are particulate matter. The estimated emissions for the industry were allocated to individual plants on the basis of their share of industry employment.

EXHIBIT B.11: NON-FERROUS SMELTERS AND REFINERIES



B.8 MOTOR VEHICLE INDUSTRIES

We examined four components of the motor vehicle industry:

- . Motor Vehicles and Motor Vehicle Parts and Accessories Surface Coating (SIC-3231);
- . Motor Vehicle Steering and Suspension Parts (SIC-3254);
- . Brake-linings and Aluminum Wheels (SIC-3255); and
- . Wet Cell Motor Vehicle Battery Production (SIC-3259).

For SIC codes 3231, 3255 and 3259 the definition we use for this study differs from that used by Statistics Canada. Our analysis is restricted to the segment of each industry that emits contaminants of concern to this study. The establishments in these four industries were obtained from the Made in Ontario and Statistics Canada data sets. The specific establishments of interest were identified in consultation with Senes and an expert on the Ontario motor vehicle industry. Emissions are apportioned on the basis of the number of employees per establishment within each industry.

The motor vehicle industry is situated primarily in southwestern Ontario (see Exhibit B.12 and B.13). This analysis includes 255 establishments with 134,993 workers.

The surface coating operations are the major source of contaminants for the motor vehicle and parts and accessories industries. Our list identifies 212 plants with 125,769 workers that have surface coating facilities. The major contaminants emitted by the surface coating activities are volatile organic compounds.

We identified 32 motor vehicle steering and suspension parts establishments (7,106 workers) and 6 brake lining and aluminum

EXHIBIT B.12: AUTOMOTIVE INDUSTRY

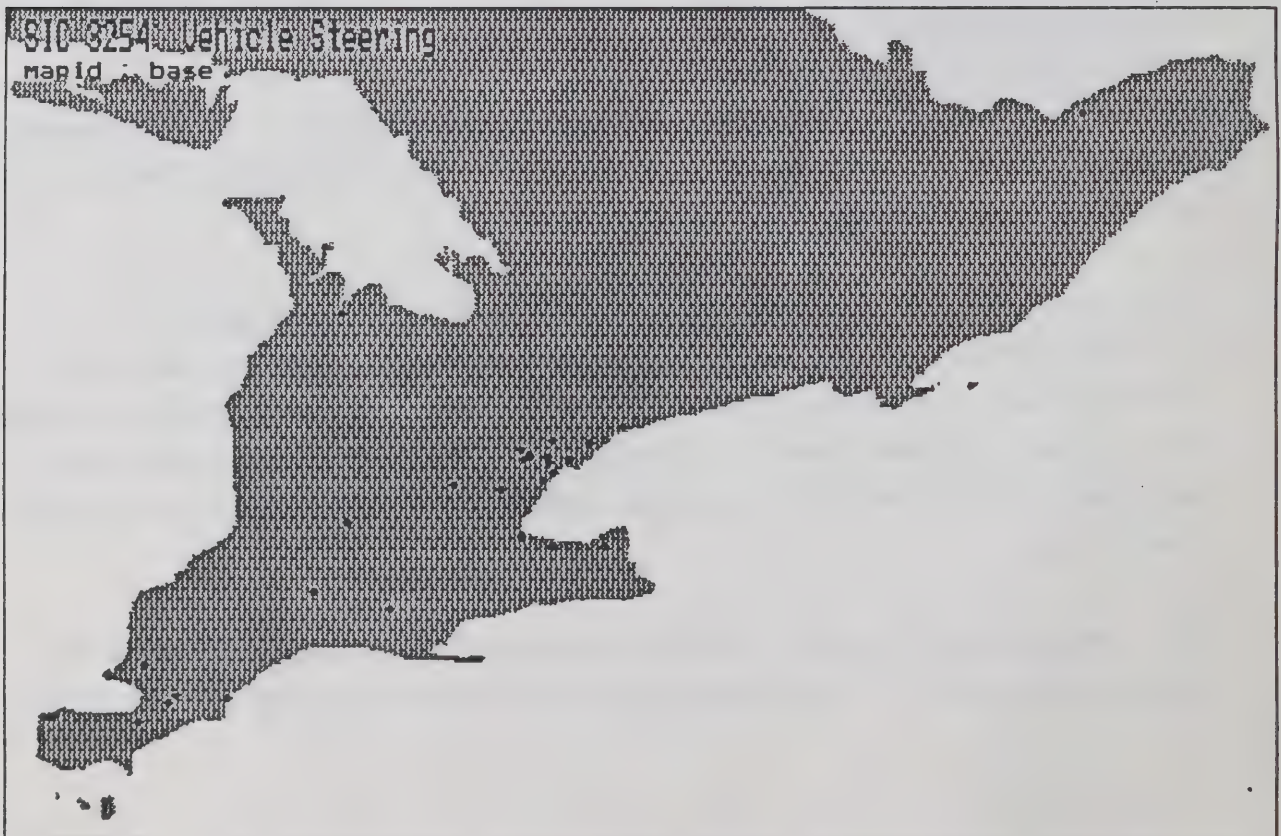
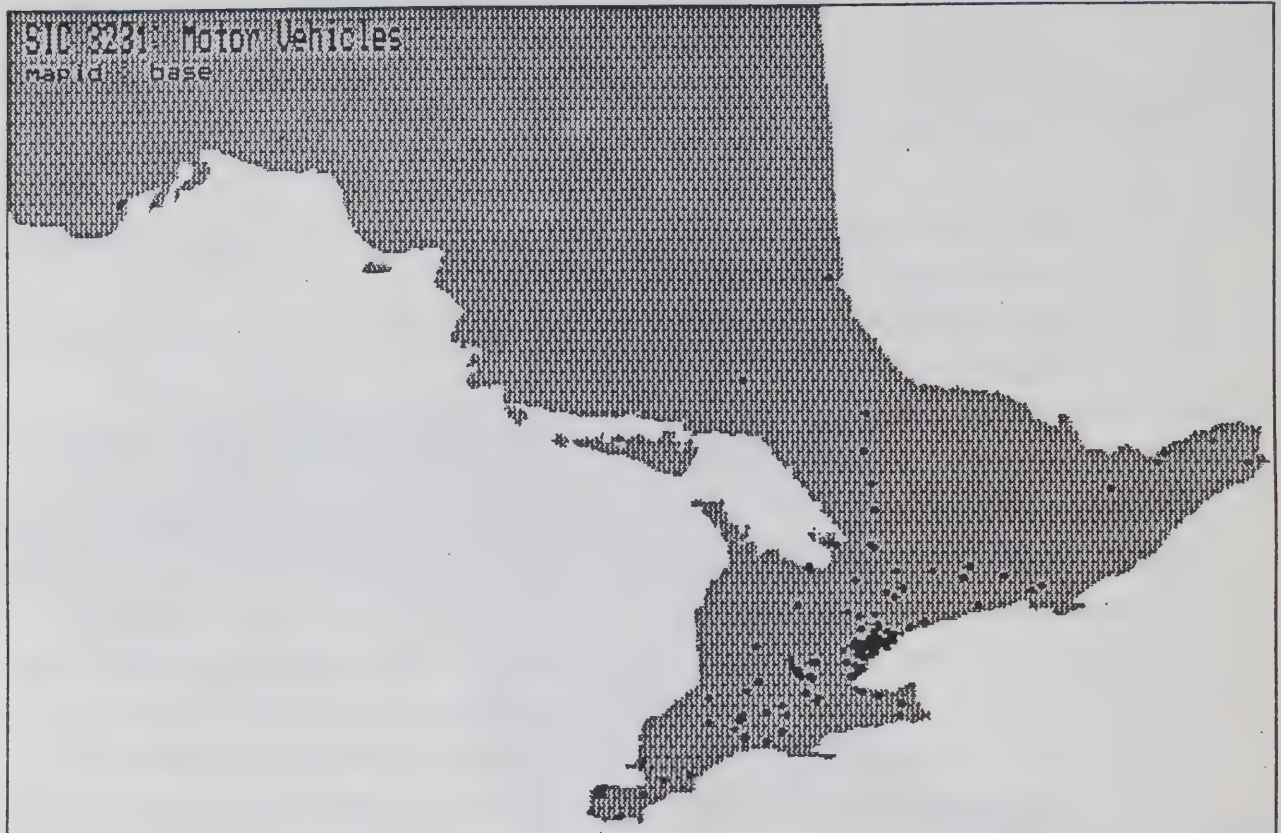
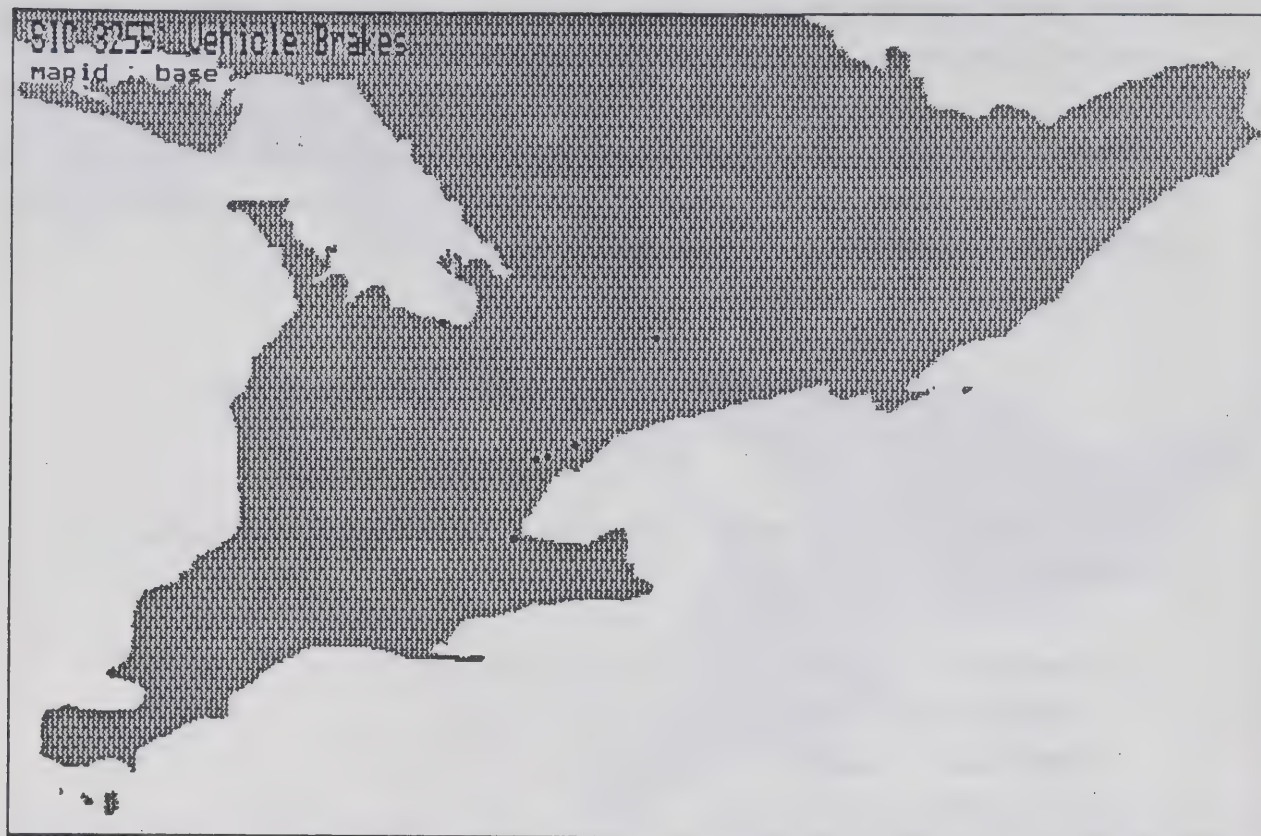


EXHIBIT B.13 : AUTOMOTIVE INDUSTRY



wheel manufacturers (1,570 workers) in Ontario. Both groups emit particulate matter.

We also listed 5 motor vehicle battery manufacturers, employing 548 workers in Ontario. The major contaminants emitted are particulate matter, primarily lead.

B.9 INORGANIC CHEMICAL MANUFACTURING

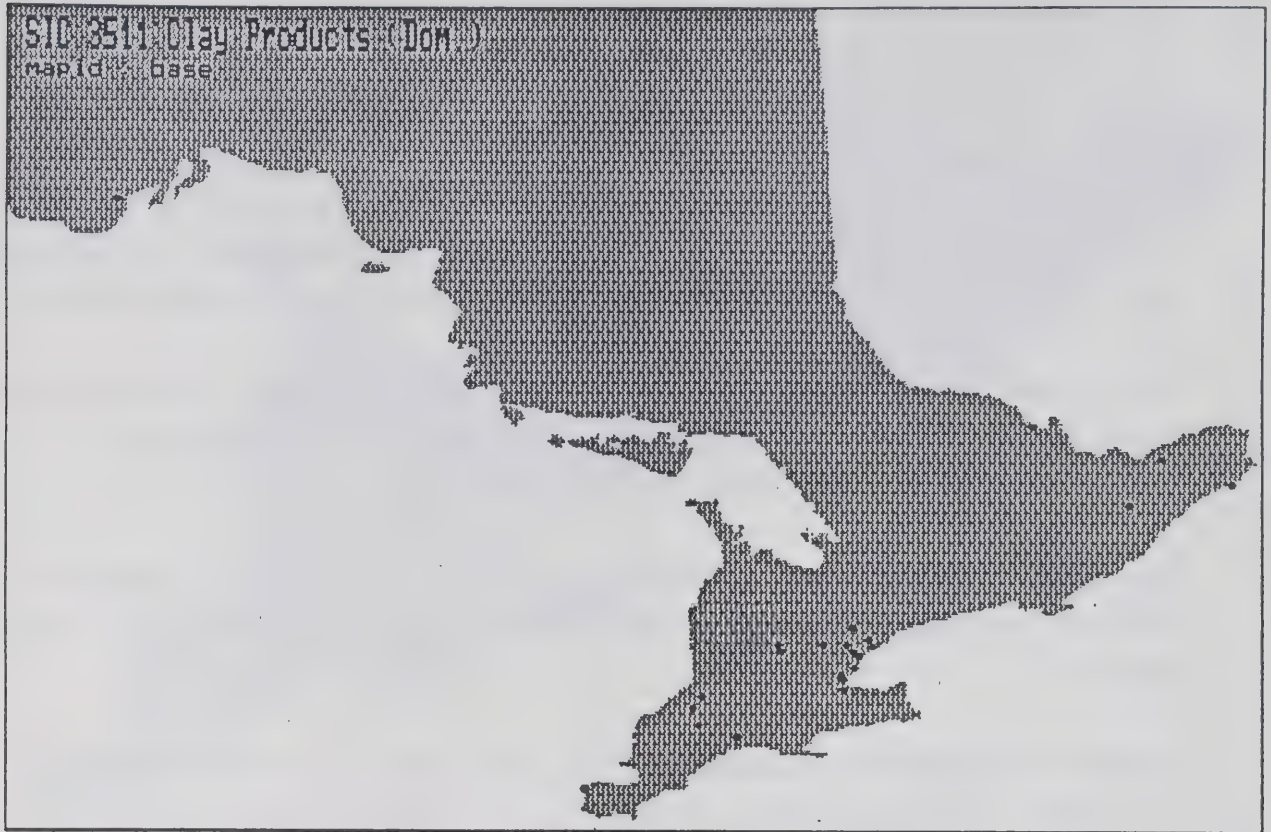
The analysis covers seven inorganic chemical manufacturing industries:

- . Domestic Clay Products (SIC-3511);
- . Imported Clay Products (SIC-3512);
- . Hydraulic Cement (SIC-3521);
- . Abrasives (SIC-3571);
- . Lime (SIC-3581);
- . Industrial Inorganic Chemicals (not elsewhere classified) (SIC-3711); and
- . Chemical Fertilizer and Fertilizer Materials (SIC-3721).

In total there are 195 inorganic chemical manufacturing establishments in Ontario employing 17,567 people. All seven industry listings were derived from the Made in Ontario data base and Statistics Canada. The establishments are concentrated in southwestern and southcentral Ontario. Individual establishment emissions are determined on the basis of plant employment relative to industry employment.

The 26 domestic clay industries (see Exhibit B.14) employ 1,405 workers. The major contaminants emitted are SO₂, fluorides, particulate matter, and NO_x.

EXHIBIT B.14: INORGANIC CHEMICAL MANUFACTURING INDUSTRIES



The imported clay products industry employs 1,187 workers at 51 different establishments (see Exhibit B.14). Particulate matter is the major contaminant emitted.

There are 8 hydraulic cement plants employing 2,092 people in Ontario (see Exhibit B.15). The major contaminants emitted are NO_x , SO_2 , volatile organic compounds, and particulate matter.

The abrasives industry has 25 establishments and 1,906 employees (see Exhibit B.15). The major emissions of the abrasives industry are SO_2 and particulate matter.

The lime industry has nine plants and 885 workers in Ontario (see Exhibit B.16). The major contaminant emissions are particulate matter.

Inorganic chemical manufacturing not elsewhere classified includes 67 establishments and 7,905 employees engaged in the production of inorganic acids, alkalis, salts, compressed gases, radioactive chemical elements and other inorganic compounds production (see Exhibit B.16). The major contaminants emitted are NH_3 , particulate matter, SO_2 , fluorine, volatile organic compounds, and NO_x .

The chemical fertilizer and fertilizer materials industry employs 1,279 people at 13 different plant locations in Ontario (see Exhibit B.17). The primary contaminants emitted are particulate matter.

B.10 PETROLEUM REFINING INDUSTRY

A total of 7 operating petroleum refineries (SIC 3611) were identified in Ontario through the Made in Ontario data base and

EXHIBIT B.15: INORGANIC CHEMICAL MANUFACTURING INDUSTRIES

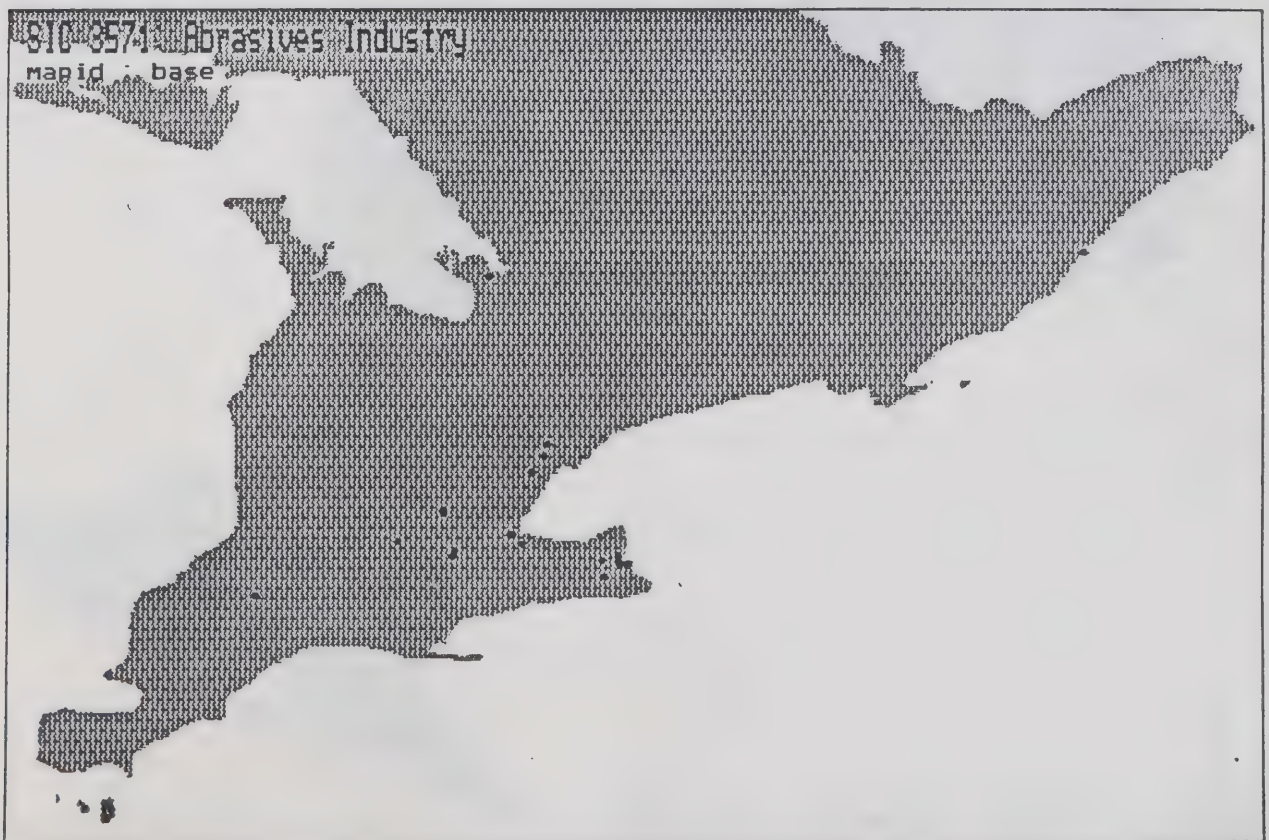


EXHIBIT B.16: INORGANIC CHEMICAL MANUFACTURING INDUSTRIES

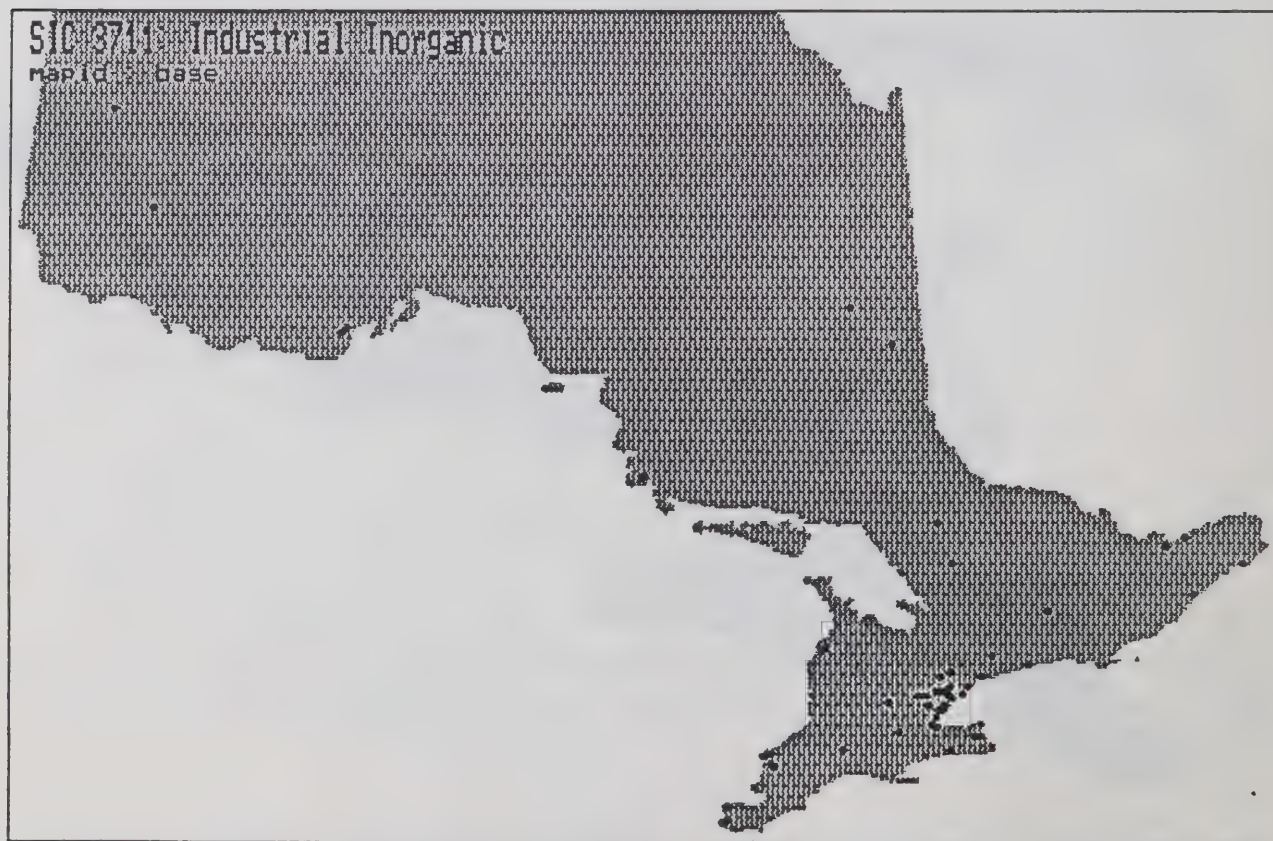
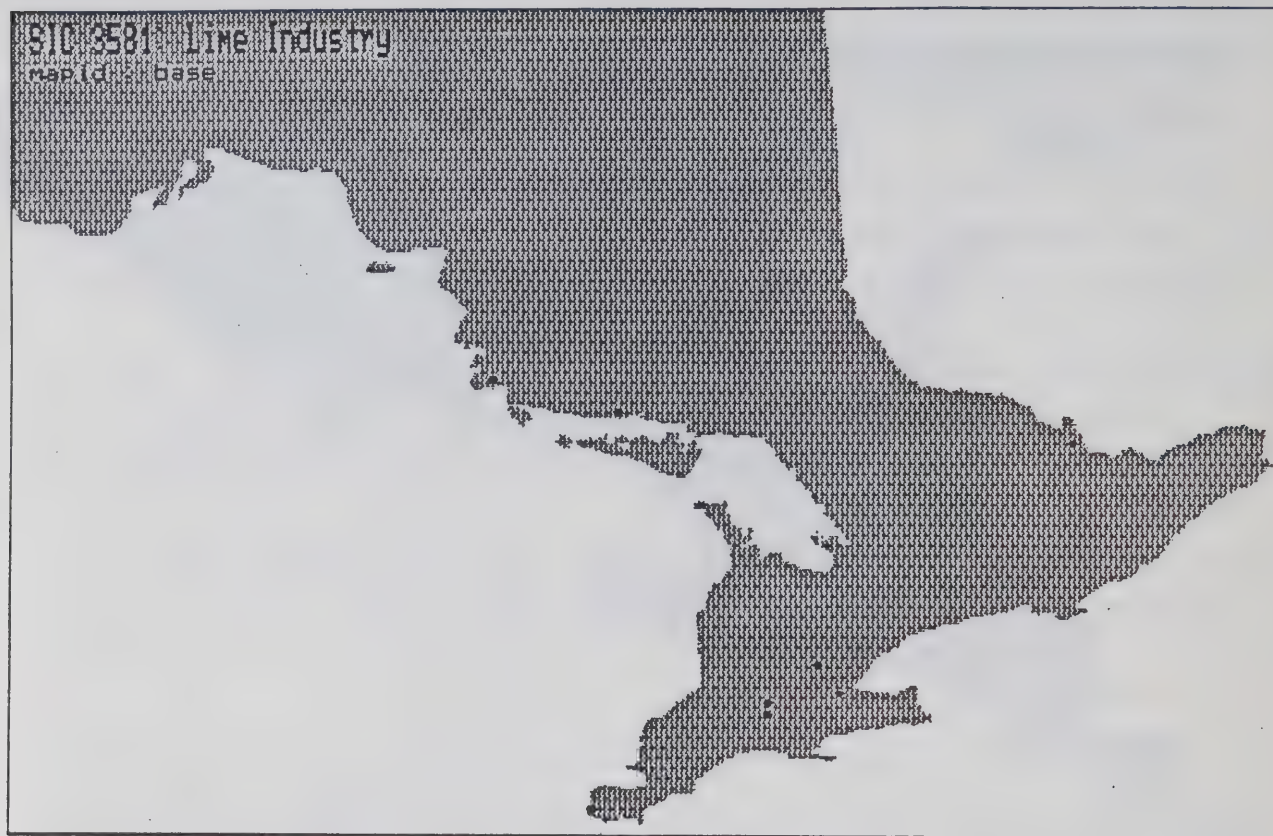
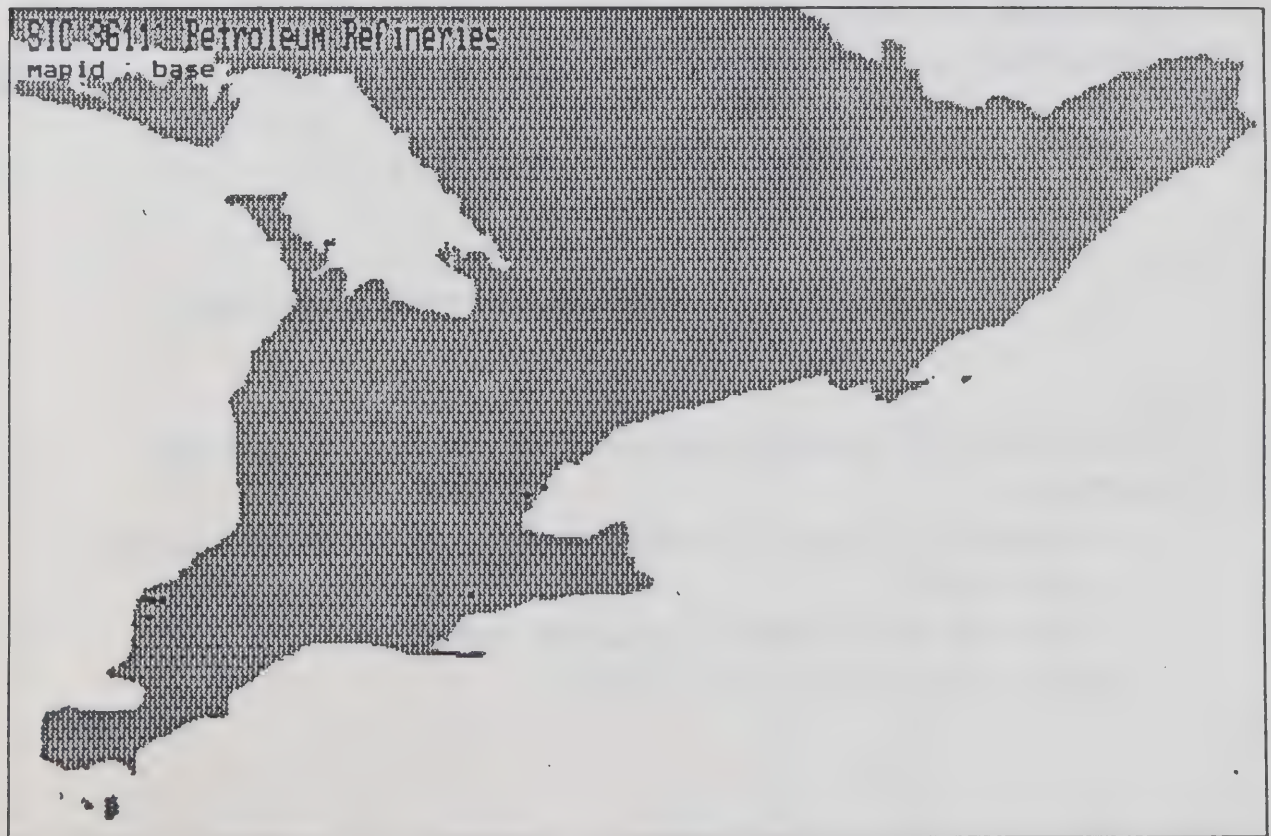
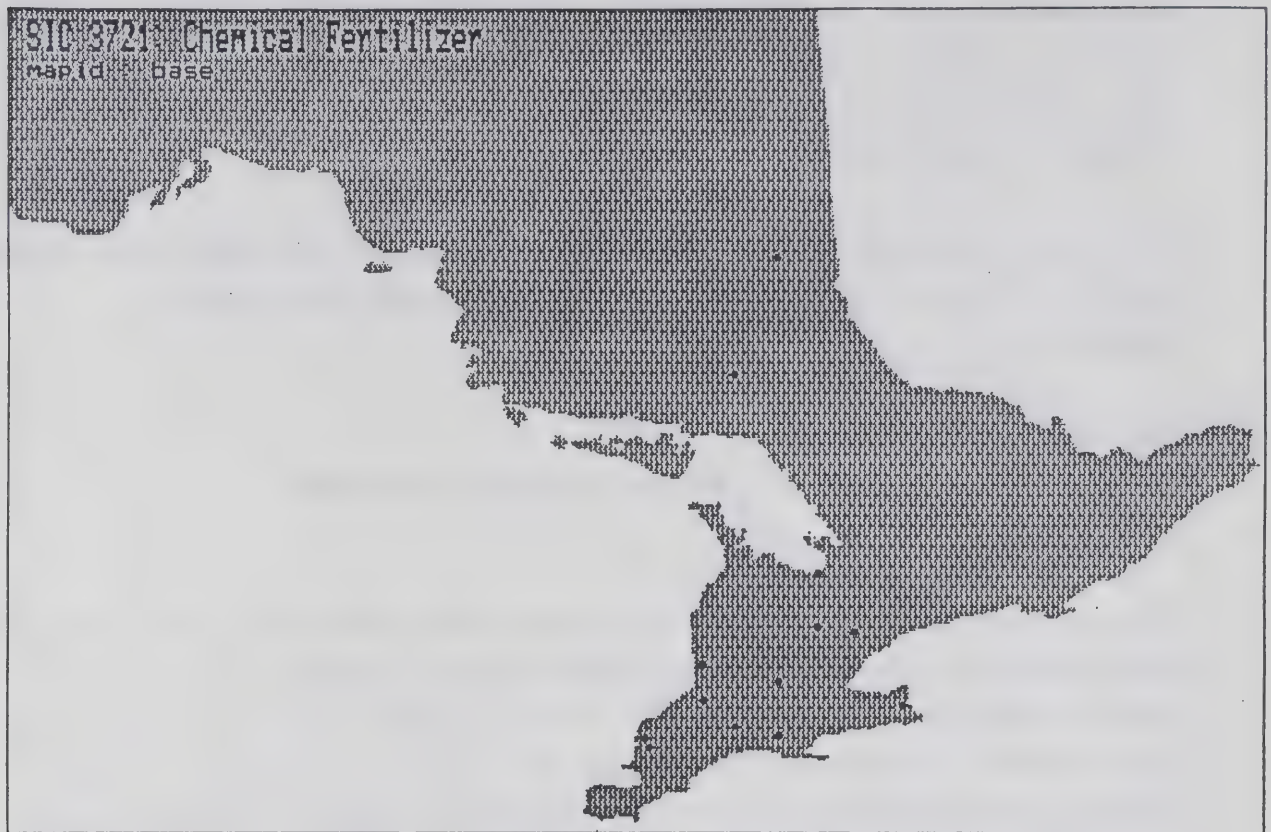


EXHIBIT B.17: CHEMICAL FERTILIZER AND PETROLEUM REFINING INDUSTRIES



consultations with Senes. These establishments are located in southwestern and southcentral Ontario (see Exhibit B.17). The number of workers employed in this industry is 3,810. Emissions are apportioned using employment at the plant in relation to industry employment.

The major contaminants emitted by the petroleum refining industry are SO₂, NO_x, particulate matter, and volatile organic compounds.

B.11 ASPHALT PAVING INDUSTRY

Our list of 24 asphalt paving establishments (SIC 3699) includes only portable plants. The estimate of the number of portable plants was provided by Senes. These plants have been randomly distributed throughout the province for the purpose of this study (see Exhibit B.18). The permanent asphalt paving plants are excluded from this study since these operations already meet Regulation 308 control standards. The portable asphalt contaminant emissions are distributed evenly among the 24 plants.

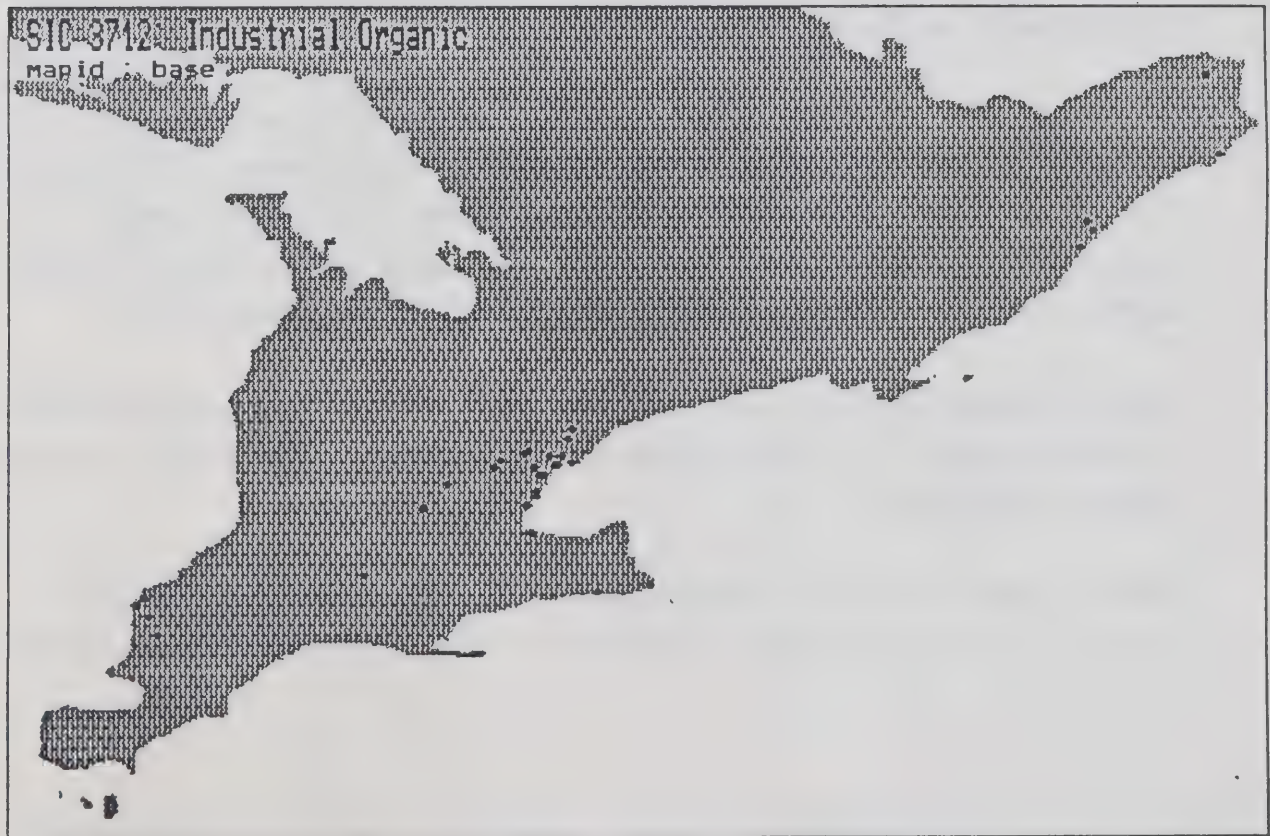
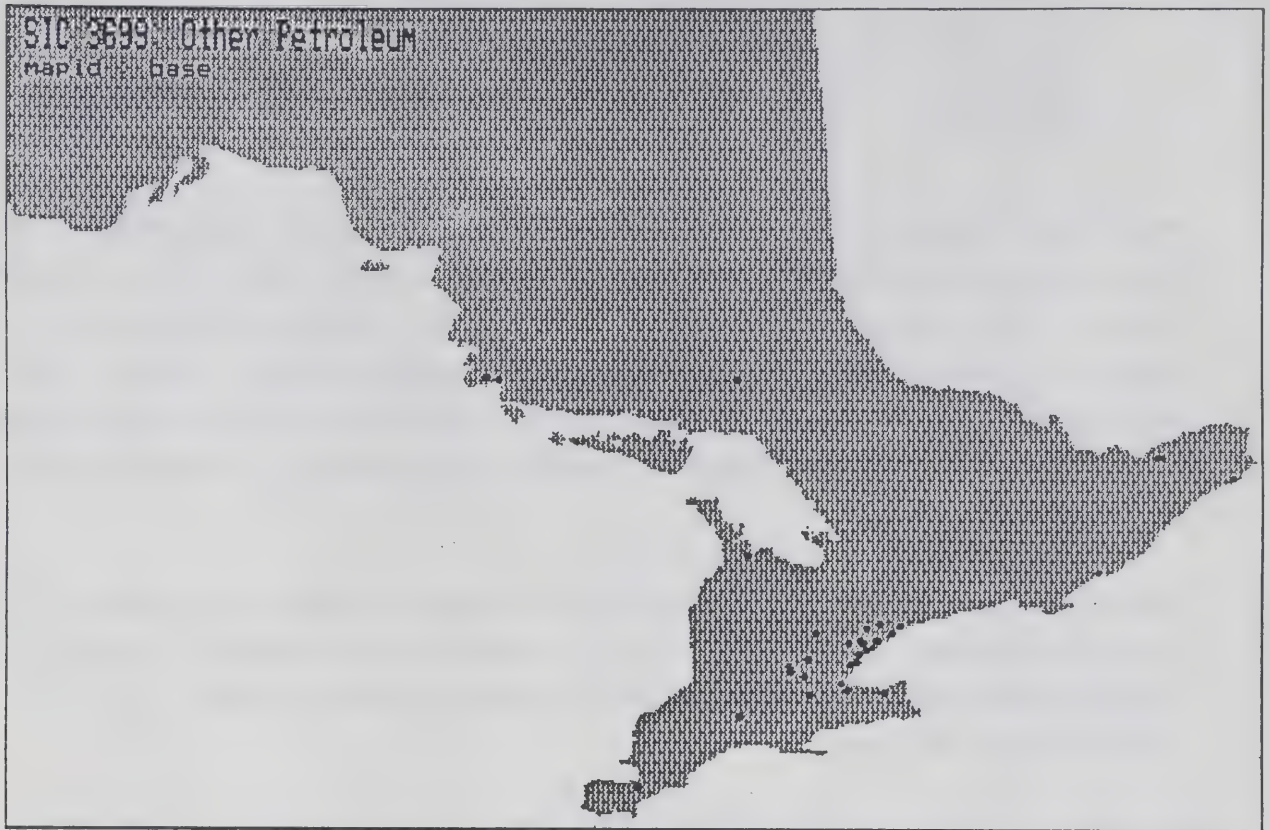
The major emissions are particulate matter.

B.12 ORGANIC CHEMICAL PRODUCTS INDUSTRY

Our analysis of organic chemicals covers the following industries:

- . Industrial Organic Chemicals (not elsewhere classified) (SIC-3712);
- . Plastics and Synthetic Resins (SIC-3731);
- . Paint and Varnish (SIC-3751);

EXHIBIT B.18: ASPHALT PAVING AND ORGANIC CHEMICALS PRODUCTS INDUSTRY



- . Printing Ink (SIC-3791);
- . Adhesives (SIC-3792); and
- . Other Chemical Products (not elsewhere classified) (SIC-3799).

The list includes 444 establishments and 30,455 employees. The data sources used are the Made in Ontario and Statistics Canada lists. The plants are located primarily in the southcentral region of the province, particularly metropolitan Toronto (see Exhibits B.18, B.19, B.20 and B.21). Emissions by establishment are estimated on the basis of plant employment in relation to total employment.

There are a total of 37 industrial organic chemicals producing establishments, employing 7,114 workers in Ontario. The major contaminants emitted are volatile organic compounds, NO_x, and particulate matter.

We list 5,539 employees working at 80 different plants in the plastic and synthetic resins industry in Ontario. The primary contaminant emitted is styrene.

The paint and varnish industry in Ontario is comprised of 98 establishments with a total of 6,185 employees. The printing ink industry is comprised 40 plants with 1,693 employees. Volatile organic compounds and particulate matter are the most common contaminants emitted by both of these industries.

The adhesives industry has a total of 1,469 employees working at 43 establishments. The major contaminants emitted are volatile organic compounds.

Lastly, there are 188 establishments which produce chemical products not elsewhere classified. Total employment is listed as

EXHIBIT B.19: ORGANIC CHEMICAL PRODUCTS INDUSTRY

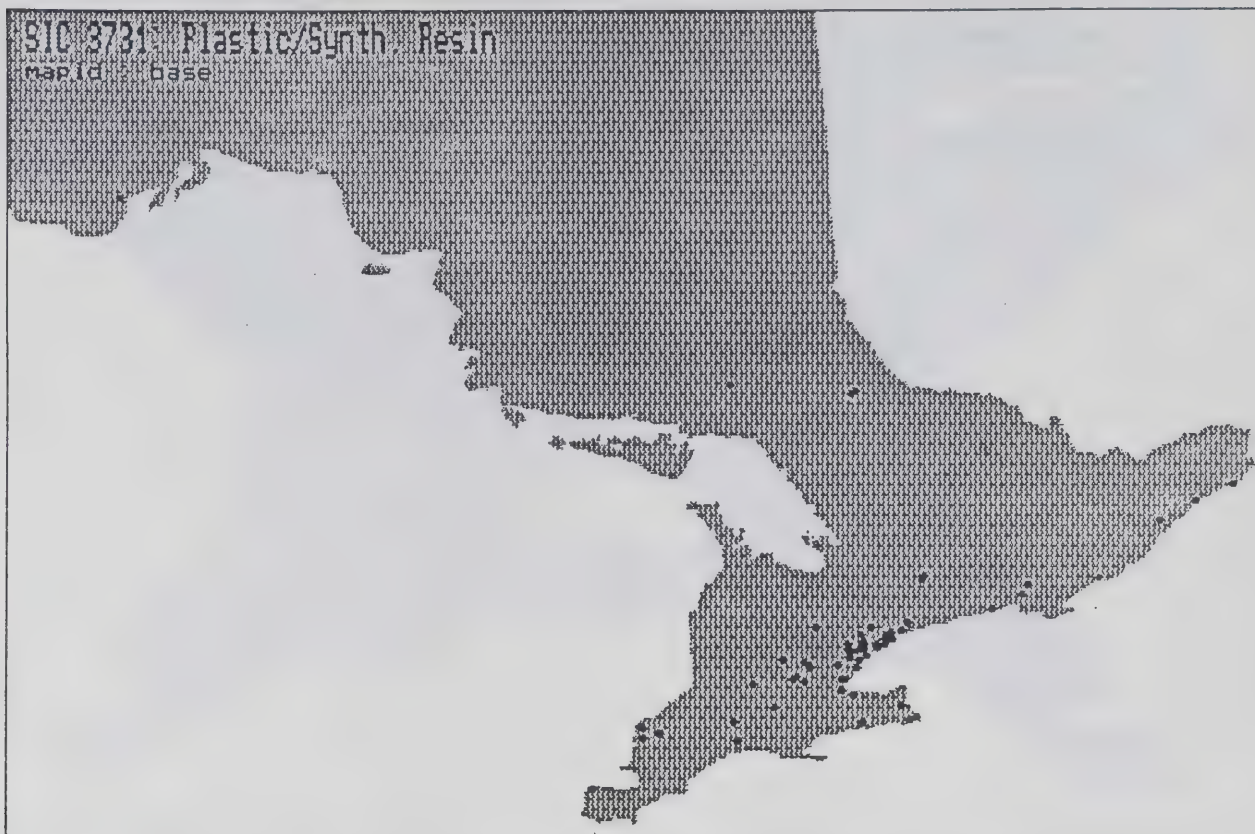
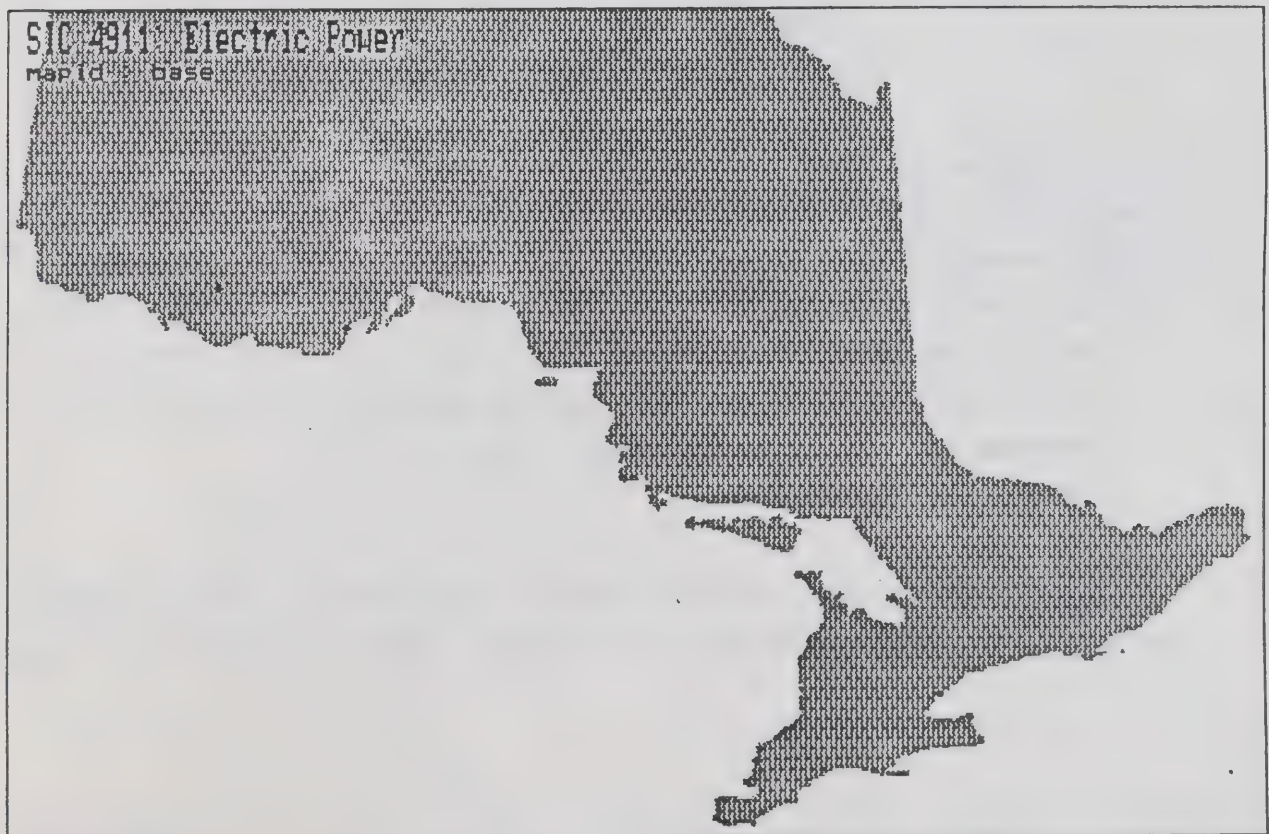
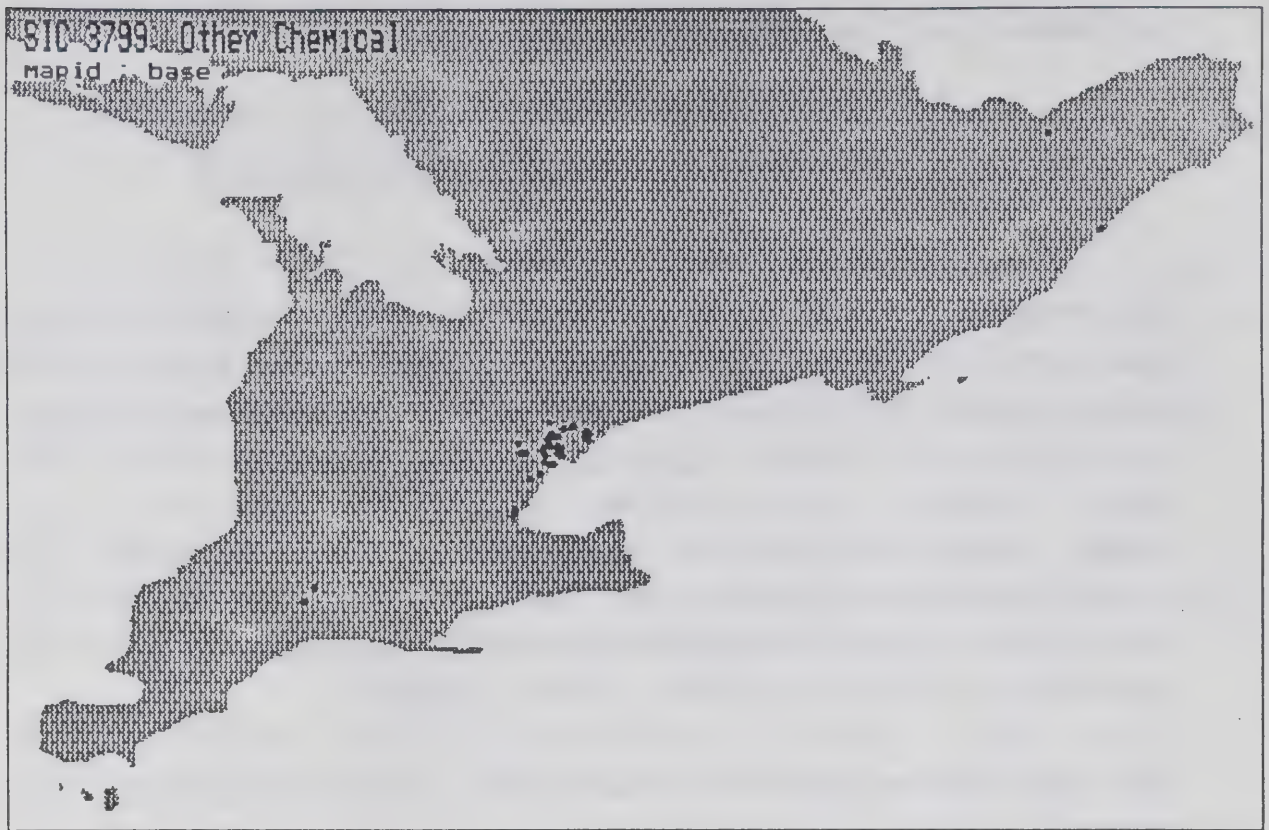


EXHIBIT B.20.: ORGANIC CHEMICAL PRODUCTS INDUSTRY



EXHIBIT B.21: ORGANIC CHEMICAL PRODUCTS AND ELECTRIC POWER INDUSTRIES



9,891. The major contaminants emitted are volatile organic compounds, particulate matter, NO_x and lead.

B.13 ELECTRIC POWER SYSTEMS INDUSTRY

The analysis covers electric power generating plants (SIC 4911) that burn coal or heavy oil. This excludes natural gas, nuclear and hydro-electric generating systems. The list was obtained from Statistics Canada, Electric Power Statistics, 1987. In total 6 electric power generating plants are included in the study. They are primarily situated in or near the major urban areas in southern Ontario (see Exhibit B.21). Emissions are distributed among the establishments on the basis of production capacity as a share of total rated capacity.

The contaminants emitted by the power plants are particulate matter.

B.14 WASTE DISPOSAL INDUSTRIES

A total of 155 establishments are identified as part of the Ontario waste disposal industry (SIC-4999). This includes:

- . Municipal Land Fill Sites;
- . Municipal and Commercial Incinerators;
- . Sewage Sludge Incinerators; and
- . Hospital Incinerators.

The locations of all waste disposal facilities except hospital incinerators were obtained from Senes. The locations of hospital

incinerators were obtained from the Ministry of the Environment (see Exhibit B.22).

There are 47 municipal land fill sites identified in Ontario (see Exhibit B.22). The major contaminant emitted is ammonia.

This study covers two municipal incinerators and one commercial incinerator (see Exhibit B.23). The contaminants emitted by the commercial and municipal incinerators are particulate matter, SO₂, NO_x, hydrogen chloride, lead, mercury, cadmium, chromium, PCDD, PCDF and PCB.

There are five sewage sludge incinerators in Ontario (see Exhibit B.23). The contaminants that they emit are particulate matter, SO₂, NO_x, hydrogen chloride and lead.

We identified hospital incinerators in Ontario. The major contaminants they emit are particulate matter and NO_x.

B.15 DRY CLEANERS

We used Senes' estimates of 1,200 dry cleaning establishments (SIC-9721) in Ontario. These establishments are distributed throughout the province on the basis of population concentration (see Exhibit B.24). Emissions are distributed evenly among all the establishments. The main contaminant emitted is perchloroethylene.

EXHIBIT B.22: WASTE DISPOSAL INDUSTRIES

SIC 499H: Hospital Incin s
rapid : base



SIC 499L: Landfill Sites
rapid : base

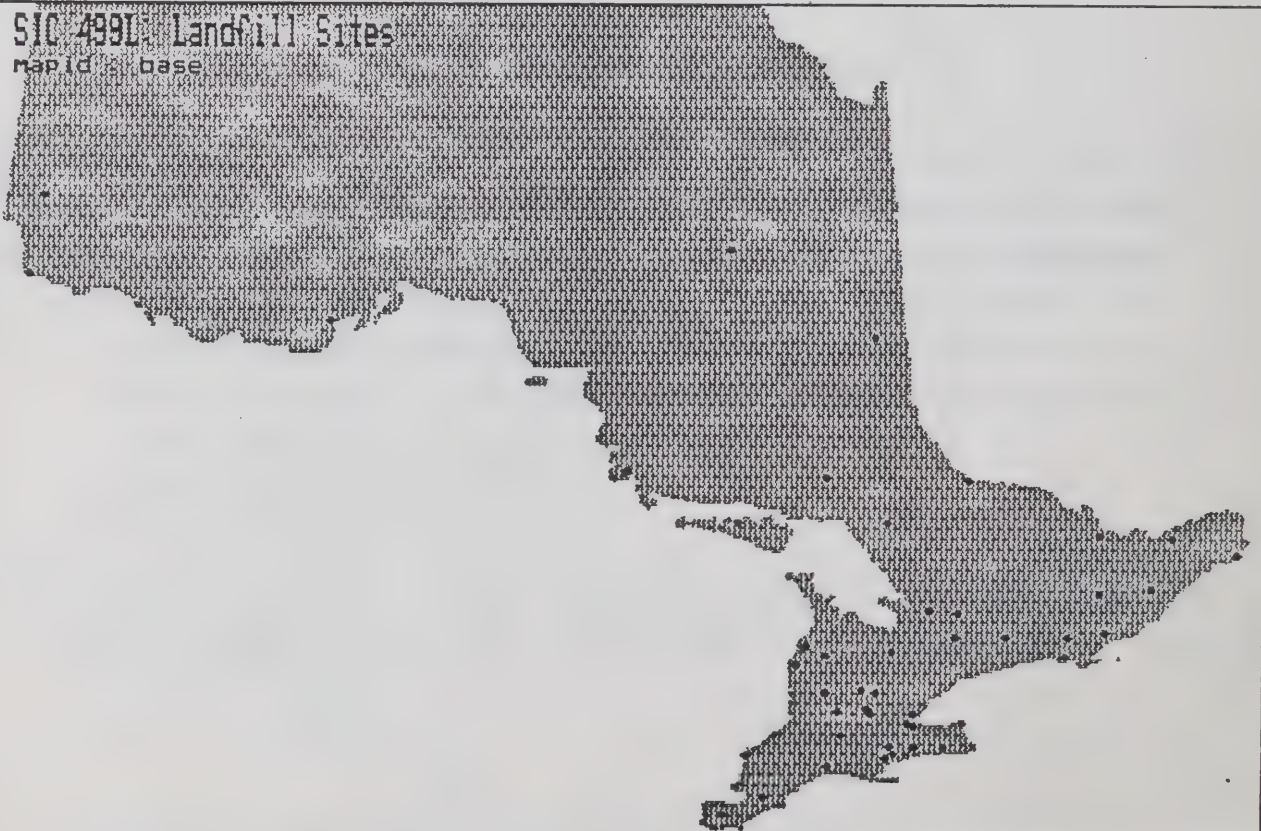


EXHIBIT B.23: WASTE DISPOSAL INDUSTRIES

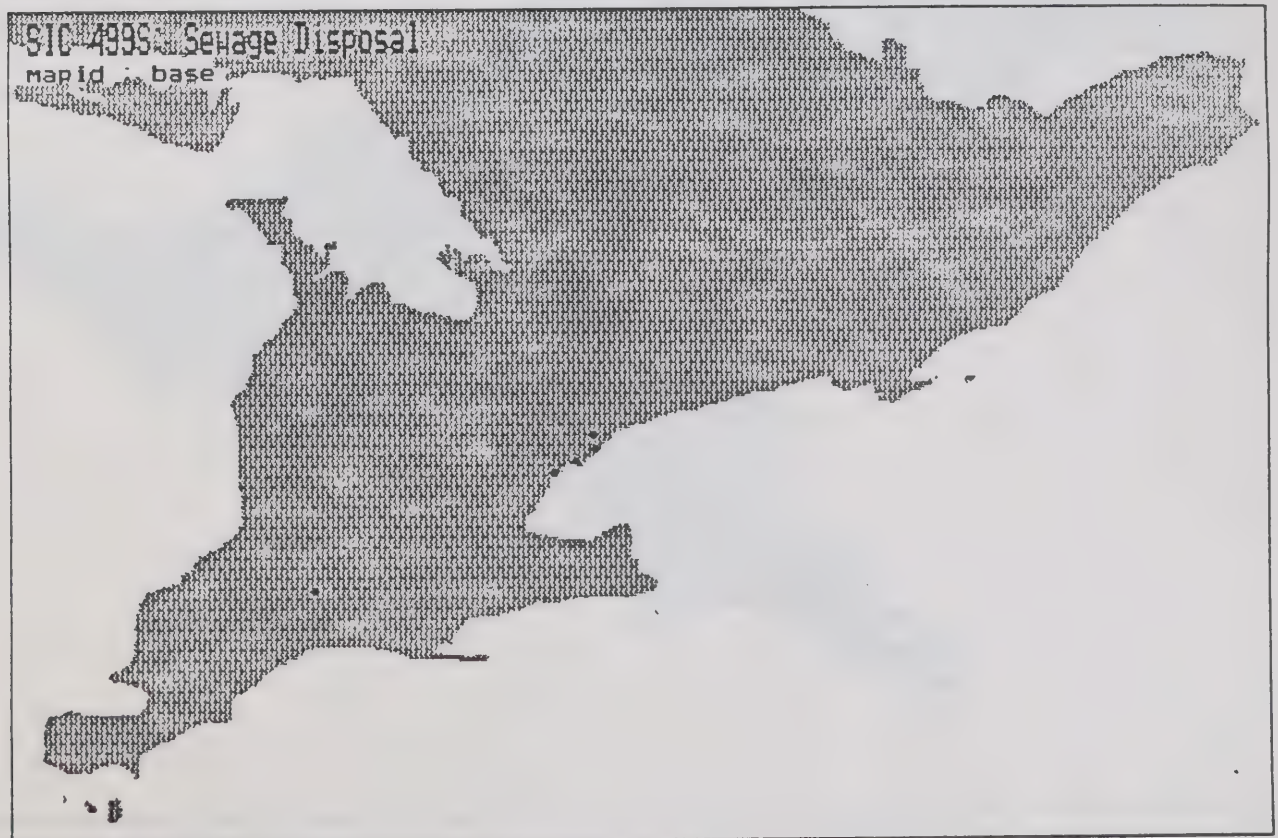
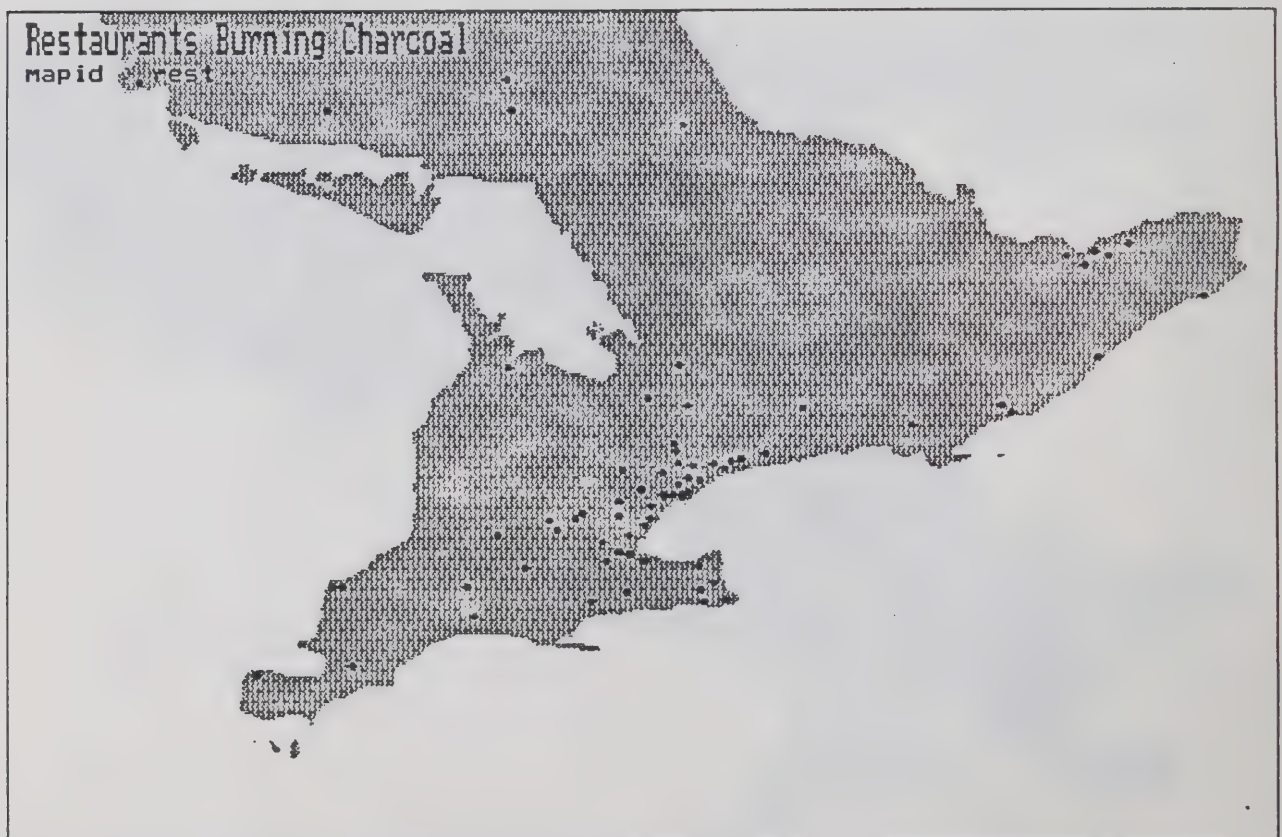
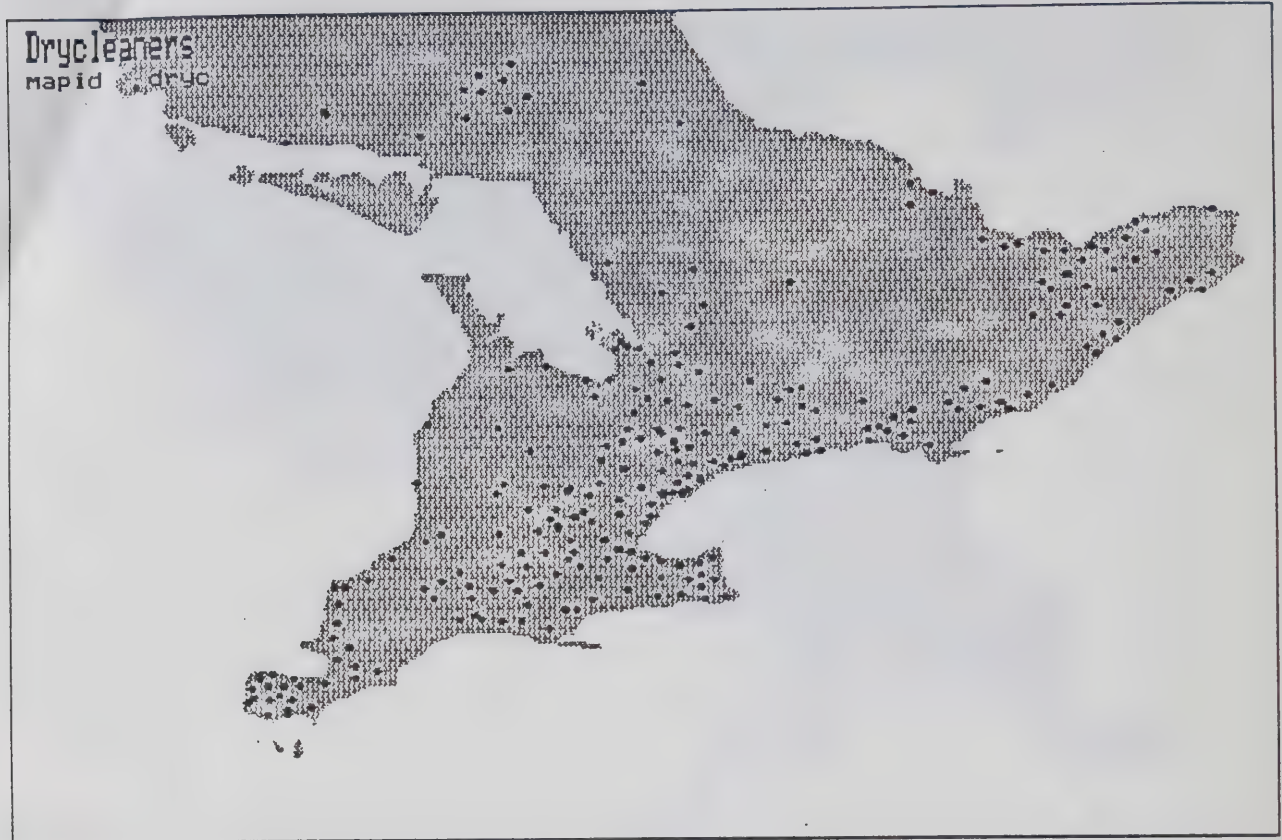


EXHIBIT B.24: DRY CLEANING AND RESTAURANT INDUSTRIES



B.16 RESTAURANTS

We used Hickling's estimate of 200 restaurants (SIC 9211-9213) that burn charcoal (barbecue) in Ontario. The establishments are distributed throughout the province on the basis of population concentration (see Exhibit B.24). The primary contaminant emitted are odour units. Emissions are evenly distributed among all restaurants.

APPENDIX C

Regulation 308 Contaminants

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C.1 INTRODUCTION

This appendix provides detailed health and environmental effects profiles for most of the 96 Regulation 308 Contaminants evaluated in this study.

Some of the chemicals listed here are proxies for aggregations of chemicals which constitute Regulation 308 Contaminants. For example, 2,3,7,8-Tetrachlorodibenzo-p-dioxin is used to represent chlorinated dibenzo dioxins and chlorinated dibenzo furans. Other chemicals cover two or more similar chemicals which are separate in the list of 96 Regulation 308 Contaminants. For example, chromium VI covers dichromium and different valencies of chromium itself.

Health and environmental effects evaluation is based on unit risk factors, threshold values, and exposure-response functions contained in the following chemical profiles.

Data presented in the chemical profiles were developed during a search of MOE chemical dossiers, peer review and government literature, and on-line data bases. Because of the large number of chemicals and effects categories evaluated, data quality varies considerably across the profiles.

Generally, there is considerably more data available for human health effects than other effects categories considered in the report. Even so, data characterizing human health response to air pollutants is typically very sparse, and in many cases is based only on very limited animal study data.

When possible, exposure-response functions have been calculated for well researched pollutants; for example, sulfur dioxide and nitrogen dioxide.

C.2 DESCRIPTION OF THE INFORMATION

There are thirteen different fields of information relating to each chemical. Eight of these represent different types of effects.

Field 1 presents the chemical name and CAS registration number.

Field 2 contains information about the chemical's carcinogenicity.

Field 3 contains information about the chemical's systemic human health effects.

Field 4 presents a list of air, and in some cases water, quality standards promulgated by the Ontario Ministry of the Environment and the United States Environmental Protection Agency.

Field 5 lists various workplace standards proposed by the Ministry of Labor, the American Conference of Governmental Industrial Hygienists (ACGIH), the U.S. Occupational Safety and Health Administration, and the U.S. National Academy of Sciences.

Field 6 lists various 1987 ambient air level standards for toxic air pollutants promulgated by different states within the United States. These ambient air levels have been established to provide sufficient protection to the general population from harmful exposures of toxic air pollutants.

Field 7 lists animal effects, not including effects to laboratory animals used in experiments to evaluate

potential human health risks. The focus of this field is on effects that could be expected to occur to animals exposed to toxic air pollutants in agricultural, domestic, or wild settings.

Field 8 lists phytotoxic effects and thresholds.

Field 9 lists effects to aquatic ecosystems.

Field 10 lists effects to surface water bodies.

Field 11 lists visibility effects.

Field 12 lists odour thresholds.

Field 13 lists damages to man-made materials.

C.3 HEALTH EFFECTS DATA SOURCES

C.3.1 Cancer Mortality

Excess cancer mortality is generally predicted using unit risk factors calculated by the U.S. EPA's Cancer Assessment Group and contained in the Integrated Risk Information System (IRIS) data base. Unit risk factors developed by the State of Massachusetts are used for a limited number of chemicals.

C.3.2 Systemic Health Effects

Systemic effects are characterized by using a number of quite different data sources. Principal sources include the MOE's dossier, which were made available to DPA staff; the U.S. Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profiles; the U.S. EPA's Baltimore Integrated Environmental Management Project (IEMP); and the U.S. EPA's oral risk reference dose (RfD) values contained in IRIS¹. ATSDR data include calculated minimal risk levels (MRLs) for acute, intermediate, and chronic exposures. Exposures below calculated MRLs are considered protective of the general population's health. The Baltimore IEMP study calculated exposure thresholds for different health endpoints. Exposures above these values are considered potentially harmful to human health. The IRIS RfDs estimate maximum safe daily lifetime oral chemical exposures. Conversion of these oral RfDs to represent inhalation exposures is highly questionable, and the U.S. EPA is currently developing separate inhalation exposure RfDs.

¹ Oral RfDs were converted from units expressing mg/kg/day to ug/m³ by (mg/kg/day)/2.86 x 10⁻⁴.

C.3.3 Proposed Regulation 308 Thresholds

In the most cases, the proposed Regulation 308 ambient air quality regulations have been set to protect human health. In these cases, these values have been used as thresholds for estimating potential human health risks due to toxic air pollutant emissions.

C.3.4 Workplace Air Quality Standards

For the purposes of comparison, numerous workplace and U.S. state ambient air levels have been listed as well: see fields 5 and 6 respectively. Workplace place standards have been developed to protect workers from potentially hazardous exposures to toxic air pollutants. These standards are not considered protective of the general or sensitive populations, and have not been developed for continuous exposures.

C.3.5 Ministry of Labour Exposure Values

The Ministry of Labour publishes three exposure values for airborne chemicals:

1. The time-weighted average exposure value (TWAEV) is the average concentration to which a worker is exposed in a work day of a work week.
2. The short term exposure value (STEV) is the maximum concentration to which a worker may be exposed in any 15 minute period determined from a single sample or a time weighted average of sequential samples taken during such periods.
3. The ceiling exposure value (CEV) is the maximum concentration to which a worker may be exposed at any time.

C.3.6 ACGIH Thresholds

The ACGIH publishes three threshold limit values:

1. The TLV-TWA is defined as the time weighted average concentration limit for a normal 8 hour workday and 40 hours per week, to which nearly all workers may be repeatedly exposed, day after day, without adverse effect.
2. The TLV-STEL is a 15-minute time-weighted-average concentration for a normal 8 hour workday and 40 hour workweek. All workers should be able to withstand up to 4 exposures per day of concentrations as high as the TLV-STEL with no ill effects if the TLV-TWA is not also exceeded.
3. The TLV-C is the airborne concentration that should not be exceeded in the workplace under any circumstances.

C.3.7 Other Workplace Thresholds

The National Academy of Sciences has developed Emergency Exposure Guidance Levels (EEGL), defined as a concentration of a substance in air judged by the U.S. Department of Defense to be acceptable for the performance of specific tasks for military personnel during emergency conditions lasting 1-24 hours. Exposure to an EEGL is not considered safe, but acceptable during tasks to prevent greater risks, eg., fires or explosions. The Academy has also developed Short-term Public Exposure Guidelines (SPEGLs), defined as acceptable levels for a single, unpredicted short-term exposure to the public. The exposure period is usually less than 1 hour, but never greater than 24 hours.

OSHA has developed Permissible Exposure Limits (PELS) as workplace exposure standards. Most of the PELs have been based on ACGIH TLVs, and adopted in 1971. The National Institute for Occupational Safety and Health (NIOSH) developed immediately dangerous to life and health (IDLH) values for approximately 390 chemicals. The IDLH concentration represents the maximum concentration of a substance in air from which healthy male workers can escape without loss of life or irreversible health effects under conditions of a maximum 30 minute exposure period.

C.3.8 State Ambient Air Levels

Ambient Air Levels (AALs) have been developed by state governments in the United States to protect their populations from harmful exposures to toxic air pollutants. AALs are derived using a variety of methodologies and are expressed using different averaging times from state to state. Consequently, there is considerable variation in the protection afforded by different AALs. In many cases, AALs are lower (i.e., the standard is more conservative) than the corresponding proposed Regulation 308 ambient air quality standard.

C.4 ACRYLONITRILE (107-13-1)

Cancer Effects:

Classified as a probable human carcinogen by the U.S. EPA [1]. Basis is the observation of a statistically significant increase in lung cancer incidence in exposed workers and observation of tumors in studies in two rat strains exposed by various routes. Classified as a 2A carcinogen by IARC, A2 by MAK, b by NTP, A2 by ACGIH, and as a carcinogen by NIOSH [2]. IRIS inhalation slope factor-- $2.4E-1$ mg/kg/day IRIS inhalation unit risk-- $6.8E-5$ ug/cu.m U.S. EPA estimates that maximum individual risk of cancer is $3.8E-3$ in the U.S.

Non-Cancer (Systemic) Effects:

Readily absorbed from the respiratory system, acrylonitrile is very toxic. U.S. EPA estimated probable oral lethal dose is 50-500 mg/kg for 70 kg person. Toxic concentrations reported at 16 ppm/20 minutes (34.4 mg/cu.m/20 min.). Acute toxicity similar to cyanide poisoning. Inhalation or ingestion results in collapse and death due to tissue anoxia and cardiac arrest.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 300 ug/cu.m

MOE (Standard) 24 hr. avg. 100 ug/cu.m

Prop. 308 24 hr. avg. 100 ug/cu.m

Water:

U.S. Clean Water Act

U.S. Ambient Water Quality Criteria

Water and Fish Consumption $5.8E-2$ ug/L

Fish Consumption Only $6.5E-1$ ug/L

Acute LEC $7.55E+3$ ug/L

Chronic LEC $2.6E+3$ ug/L

Advisories:

MOL TWAEV 2 ppm (4.3 mg/cu.m)

MOL STEV 10 ppm (21.7 mg/cu.m)

ACGIH TLV-TWA 2 ppm (4.5 mg/cu.m)

IDLH 500 ppm

OSHA TWA 2ppm

OSHA-C 10ppm

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	22.0000	ug/cu.m	8 hr.	200.0
IN	0.0147	ug/cu.m	annual	0.0
MA	0.1500	ug/cu.m	24 hr.	0.0
NC	0.1450	ug/cu.m	24 hr.	200.0
NV	0.1070	mg/cu.m	8 hr.	42.0
NY	15.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	11.3000	ug/cu.m	1 yr.	200.0
SD	22.0000	ug/cu.m	8 hr.	200.0
VA	45.0000	ug/cu.m	24 hr.	100.0

Animal Effects:

Central nervous system effects in rats chronically exposed to 80 ppm [3].

Phytotoxic Effects:

Pea seedlings showed toxic response at 9 ppm in water--no effect seen at lower levels [4].

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 0.0031 ppm (0.0067 mg/cu.m) to 50.4 ppm (108.4 mg/cu.m); average 18.6 ppm (40 mg/cu.m) Slight odor of peach pits. 1.7-23 ppm; PIT50% 21.4% [3]. Mild, not unpleasant odor at 21.6 ppm (46.4 mg/cu.m) [5]. Odor threshold 21.4 ppm (46 mg/cu.m) [6].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.5 AMMONIA (7664-41-7)

Cancer Effects:

Not considered carcinogenic [7].

Non-Cancer (Systemic) Effects:

High concentrations cause blindness and intolerable irritation. NIOSH cites references indicating that concentrations as low as 50 ppm (34 mg/cu.m) are moderately irritating and disagreeable [7].

Standards:

Prop. 308 1 hr. avg. 3000 ug/cu.m

Advisories:

MOL TWEAV 25 ppm (17 mg/cu.m)
 MOL STEV 35 ppm (24 mg/cu.m)
 ACGIH TLV-TWA 25 ppm (18 mg/cu.m)
 ACGIH STEL 35 ppm (27 mg/cu.m)
 OSHA PEL 35 mg/ cu.m
 EEGL/SPEGL 100 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	360.0000	ug/cu.m	8 hr.	50.0
KS	42.8750	ug/cu.m	annual	20.0
MA	24.0000	ug/cu.m	24 hr.	0.0
NV	0.4290	mg/cu.m	8 hr.	42.0
NY	360.0000	ug/cu.m	1 yr.	0.0
SD	360.0000	ug/cu.m	8 hr.	50.0
VA	250.0000	ug/cu.m	24 hr.	60.0
WY	429.0000	ug/cu.m	1 hr.	42.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

10E-3.5-10E.5 mg/cu.m [8].

Complaint level 20-25 ppm (13.6-17 mg/cu.m) [7].

Odor threshold 46.8 ppm (31.8 mg/cu.m) [9].

Odor threshold 3.70E-2 mg/L [10].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.6 ARSENIC (7440-38-2)

Cancer Effects:

Classified as a human carcinogen by the U.S. EPA [11] and [12]. Basis of observation is lung cancer mortality in populations exposed through inhalation and increased skin cancer incidence in several populations consuming drinking water with high arsenic concentrations. IRIS inhalation slope factor-- $1.5E+1$ mg/kg/day IRIS inhalation unit risk-- $4.29E-3$ ug/cu.m Taking mortality due to lung cancer to be critical endpoint, and assuming that $10E-6$ to $10E-5$ cases per year is acceptable, ambient air containing 0.1 ug/cu.m to 1.0 ug/cu.m is considered acceptable [13]. Recent estimates suggest that cancer risk levels of $10E-5$ and $10E-6$ are associated with 0.001 ug/cu.m to 0.0001 ug/cu.m [14]. Classified as a 1 carcinogen by IARC, A1 by MAK, a by NTP, and as a carcinogen by NIOSH [2].

Non-Cancer (Systemic) Effects:

Primary non-carcinogenic health risk is irritation of the skin and mucous membranes. Chronic exposure of workers to 0.1 mg/cu.m may be associated with risk of some systemic effects, as well as irritation of skin and mucous membranes. ATSDR [14] estimated minimal risk levels (MRLs) for arsenic inhalation are: acute-- 1.0 ug/cu.m intermediate-- 1.0 ug/cu.m chronic-- 1.0 ug/cu.m. U.S. EPA Baltimore IEMP Threshold Value: nonspecific cellular effects-- 12.3 ug/cu.m.

Standards:

Air:

MOE (Ten. Des. Std.) 0.5 hr. avg. 1 ug/cu.m
 MOE (Ten. Des. Std.) 24 hr. avg. 0.3 ug/cu.m
 Prop. 308 24 hr. avg. 0.3 ug/cu.m

Water:

U.S. Clean Water Act
 U.S. Ambient Water Quality Criteria
 Water and Fish Consumption $2.2E-3$ ug/L
 Fish Consumption Only $1.75E-2$ ug/L
 Acute LEC $3.6E+2$ ug/L AS III
 Chronic LEC $1.9E+2$ ug/L AS III

Advisories:

MOL TWEAV 10 ug/cu.m
 MOL STEV 50 ug/cu.m 15 min. avg.
 NIOSH REL (15 min.) 2 ug/cu.m
 ACGIH TLV-TWA 0.2 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	0.0500	ug/cu.m	8 hr.	200.0
MT	0.3900	ug/cu.m	24 hr.	128.0
	0.0700	ug/cu.m	annual	714.0
NC	0.2300	ng/cu.m	annual	200.0
NV	0.0050	mg/cu.m	8 hr.	42.0
NY	0.6700	ug/cu.m	1 yr.	0.0
PA-Phil.	0.0240	ug/cu.m	1 yr.	420.0
RI	0.0002	ug/cu.m	annual	0.0
VA	3.3000	ug/cu.m	24 hr.	60.0

Animal Effects:

Maximum tolerated non-lethal dose in rats corresponds to air concentration of over 40 mg/cu.m [14].

Phytotoxic Effects:

Arsenic is phytotoxic in doses greater than those found in ambient conditions.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.7 ARSINE (7784-42-1)

Cancer Effects:

Not considered carcinogenic [15].

Non-Cancer (Systemic) Effects:

Acute arsine poisoning causes rapid intravascular hemolysis with associated symptoms of: headache, dyspnea, nausea, and vomiting. Abdominal pain and jaundice appear rapidly in severe cases. 250 ppm (800 mg/cu.m) for 30 minutes is fatal; 3-10 ppm (9.6-32 mg/cu.m) causes poisonous symptoms in a few hours [15]. Inhaled arsine may be rapidly absorbed from lung tissue and oxidized to form elemental trivalent arsenic. Trivalent arsenic compounds are carcinogenic.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 10 ug/cu.m
MOE (Standard) 24 hr. avg. 5 ug/cu.m
Prop. 308 24 hr. avg. 5 ug/cu.m

Advisories:

MOL TWEAV 0.05 ppm (0.16 mg/cu.m)
ACGIH TLV-TWA 0.05 ppm (0.2 mg/cu.m)
OSHA PEL 0.2 mg/cu.m
EEGL/SPEGL 1 ppm
IDLH 6 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	1.0000	ug/cu.m	8 hr.	200.0
NV	0.0050	mg/cu.m	8 hr.	42.0
NY	0.6700	ug/cu.m	1 yr.	0.0
VA	3.3000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

Colorless gas [15].

Odor Effects:

Disagreeable garlic odor [15].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.8 ASBESTOS (1332-21-4)

Cancer Effects:

Classified as a human carcinogen [16], however, no precise statement of risk was made by Finkelstein [17]. Continuous exposure above 1 f/cu.cm causes unacceptable risks. Classified as a 1 carcinogen by IARC, A1 by MAK, a by NTP, A1 by ACGIH, and as a carcinogen by NIOSH [2]. Massachusetts unit risk-- $7.60E-3$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

Primary non-carcinogenic health risk is asbestosis. Asbestosis is a diffuse but non-uniform fibrosis of the lungs that is generally most severe in the basilar portions. The risk of developing asbestosis following 20 years of exposure to concentrations in the range up to 1-2 f/cu.cm is unlikely to substantially exceed 1% [17].

Standards:

Air:

MOE (Guideline) 0.5 hr. avg. 5 ug/cu.m
(total asbestos)

MOE (Guideline) 24 hr. avg. .04 f/cc

Prop. 308 24 hr. avg. 0.04 f/cc (>5 um length)

Prop. 308 24 hr. avg. 1.6 ug/cu.m (total asbestos)

Advisories:

MOL recommended TWA (40 hr. wk.) 0.5
fibres/cu.cm

MOL TWEAV amosite 0.5 f/cu.cm

MOL TWEAV crocidolite 0.2 f/cu.cm

MOL TWEAV other asbestos 1.0 f/cu.cm

MOL STEV amosite 2.5 f/cu.cm

MOL STEV crocidolite 1.0 f/cu.cm

MOL STEV other asbestos 5.0 f/cu.cm

ACGIH TLV-TWA amosite 0.5 f/cu.cm,
crocidolite 0.2 f/cu.cm, chrysotile
2 f/cu.cm, other forms 2 f/cu.cm

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	0.0010	ug/cu.m	8 hr.	200.0
MA	0.0000	f/cu.m	24 hr.	0.0
NC	0.0100	ug/cu.m	24 hr.	200.0
NV	0.0000			42.0
NV-L.Veg.1000	0.0000	f/cu.m	24 hr.	0.0
NY	5.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	0.0050	ug/cu.m	1 yr.	420.0
VA	2.0000	ug/cu.m	24 hr.	100.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.9 BENZENE (71-43-2)

Cancer Effects:

Classified as a human carcinogen by [19] and by the U.S. EPA [20] and a suspected human carcinogen by the ACGIH [21]. Basis of the EPA's assessment is several studies of increased leukemia incidence due to occupational exposure, increased incidence of neoplasia in rats and mice exposed by inhalation and gavage, and other supporting data. Classified as a 1 carcinogen by IARC, A1 by MAK, a 2 by NTP, A2 by ACGIH, and as a carcinogen by NIOSH [2]. IRIS inhalation slope factor-- 2.9×10^{-2} mg/kg/day IRIS inhalation unit risk-- 8.3×10^{-6} ug/cu.m Massachusetts unit risk-- 8.1×10^{-6} ug/cu.m [18].

Non-Cancer (Systemic) Effects:

Acute exposure results in irregular heart rate, dizziness, headache, nausea, and loss of consciousness. Chronic occupational exposure has resulted in reduced peripheral nerve conduction velocity, inflammation of the respiratory tract, hemorrhage in the lungs, congestion of the kidneys and cerebral edema. ATSDR MRLs [22] for benzene inhalation are: acute--0.01 ppm (0.032 mg/cu.m)
 intermediate--0.125 ppm (0.4 mg/cu.m)
 EPA Baltimore IEMP TVs:
 blood--2.45 ug/L
 fetal/developmental--41.3 ug/cu.m

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 10000 ug/cu.m
 MOE (Standard) 24 hr. avg. 3300 ug/cu.m
 Prop. 308 24 hr. avg. 3300 ug/cu.m

Water:

MOE "undefined tolerance limit" less than 100 mg/L to protect fish
 U.S. Clean Water Act
 U.S. Ambient Water Quality Criteria
 Water and Fish Consumption 6.6×10^{-1} ug/L
 Fish Consumption Only 4.0×10^{-1} ug/L
 Acute LEC 5.3×10^{-3} ug/L
 Chronic LEC None

Advisories:

MOL TWEAV 1 ppm (3.2 mg/cu.m) not to
 exceed 5 ppm (16 mg/cu.m)
 MOL STEV 15 ppm (48 mg/cu.m)
 OSHA 8 hr. TWA 10 ppm
 OSHA TLVC 25 ppm
 ACGIH TLV-TWA 10 ppm (30 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	150.0000	ug/cu.m	8 hr.	200.0
MA	1.2000	ug/cu.m	24 hr.	0.0
NC	150.0000	ug/cu.m	24 hr.	200.0
NV	0.7140	mg/cu.m	8 hr.	42.0
NY	100.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	72.0000	ug/cu.m	1 yr.	420.0
RI	0.1000	mg/cu.m	annual	0.0
VA	300.0000	ug/cu.m	24 hr.	100.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

Numerous freshwater fish LC50 data; however, because of high volatility, there is little opportunity for benzene to accumulate in aquatic ecosystems.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

Colorless gas [21].

Odor Effects:

Odor threshold 4.68 ppm (15 mg/cu.m)
 Odor threshold 8.8×10^{-3} mg/L [23].

Odor threshold 4.6 ppm (14.7 mg/cu.m) [24].
Odor characteristic of aromatic hydrocarbons.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.10 BENZO[A]PYRENE (50-32-8)

Cancer Effects:

Classified as a probable human carcinogen by the U.S. EPA [25]. However, quantitative estimates of carcinogenic risk due to inhalation exposure are not currently available. Human data linking BaP to a carcinogenic effect are lacking. However, there are many animal studies demonstrating BaP to be carcinogenic following inhalation. Classified as a 2A carcinogen by IARC, b by NTP, and A2 by ACGIH [2]. The MOE BaP Dossier [26] cited BaP as a carcinogen by all routes, and suggested a permissible exposure limit for coal tar volatiles of 0.2 mg/cu.m TWA.

Non-Cancer (Systemic) Effects:

Information on acute, intermediate and non- cancer effects due to BaP inhalation is extremely limited. No information regarding human health endpoints or threshold values is available [25, 27].

Standards:

Air:

MOE (Prov. Guid.) 1 yr. avg. 0.3 ng/cu.m
MOE (Prov. Guid.) 24 hr. avg. 1.1 ng/cu.m single source
MOE (Prov. Guid.) 1 yr. avg. 0.22 ng/cu.m single source
MOE 0.5 hr. avg. 3.3 ng/cu.m single source
Prop. 308 24 hr. avg. 1.1 ng/cu.m, 1 yr. avg
0.22 ng/cu.m (single source)
Prop. 308 1 yr. avg. 0.3 ng/cu.m (all sources)

Advisories:

OSHA PEL 0.2 ug/cu.m
ACGIH TLV-TWA 0.2 mg/cu.m
NIOSH TLV 0.1 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	0.1000	ug/cu.m	8 hr.	200.0
PA-Phil.	0.0007	ug/cu.m	1 yr.	100.0
VA	0.0000	ug/cu.m	24 hr.	100.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.11 BERYLLIUM (7440-41-7)

Cancer Effects:

Several epidemiology studies reviewed by EPA suggested a connection between beryllium exposure and lung cancer in humans, but the data are inadequate for several reasons [28]. Beryllium compounds are, however, clearly carcinogenic in animals [29]. Classified as a 2A carcinogen by IARC, A2 by MAK, b by NTP, A2 by ACGIH, and as a carcinogen by NIOSH [2]. Massachusetts unit risk-- $2.40\text{E-}3$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

The lung is the main target organ following exposure to beryllium and beryllium compounds by inhalation. This toxicity is manifest as acute pneumonitis or a more chronic form of lung disease. The EPA has developed an oral beryllium RfD of $5\text{E-}3$ mg/kg/day (17.5 ug/cu.m)a [28].

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 0.03 ug/cu.m

MOE (Standard) 24 hr. avg. 0.01 ug/cu.m

Prop. 308 24 hr. avg. 0.01 ug/cu.m

Water:

U.S. Clean Water Act

U.S. Ambient Water Quality Criteria

Water and Fish Consumption $6.8\text{E-}3$ ug/L

Fish Consumption Only $1.17\text{E-}1$ ug/L

Acute LEL $1.3\text{E+}2$ ug/L

Chronic LEL $5.3\text{E+}0$ ug/L

Advisories:

MOL TWEAV 0.002 mg/cu.m

ACGIH TLV-TWA 0.002 mg/cu.m

OSHA PEL 2 ug/cu.m

NIOSH TLV 0.5 ug/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	0.0100	ug/cu.m	8 hr.	200.0
MA	0.0042	ug/cu.m	24 hr.	0.0
NC	0.0100	ug/cu.m	24 hr.	200.0
NV	0.0001	mg/cu.m	8 hr.	42.0
PA-Phil.	0.0100	ug/cu.m	1 yr.	1.0
SD	0.0200	ug/cu.m	8 hr.	100.0
VA	0.0200	ug/cu.m	24 hr.	100.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.12 1,3-BUTADIENE (106-99-0)

Cancer Effects:

The EPA considers butadiene to be a probable human carcinogen based on animal studies [30]. IRIS inhalation slope factor-- $1.8E+0$ mg/kg/day IRIS inhalation unit risk-- $2.8E-4$ ug/cu.m Classified as an A2 carcinogen by MAK, A2 by ACGIH, and as a carcinogen by NIOSH [2].

Non-Cancer (Systemic) Effects:

Butadiene is a central nervous system depressant in high concentrations. It is irritating to skin and mucous membranes and vapors may cause dizziness or suffocation. Death can result 23 minutes after inhaling air containing 25% butadiene [30].

Standards:

Advisories:

MOE TWEAV 10 ppm (22 mg/cu.m)
ACGIH TLV-TWA 10 ppm (22 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	22000.0000	ug/cu.m	8 hr.	50.0
MA	0.0350	ug/cu.m	24 hr.	0.0
NC	110.0000	ug/cu.m	24 hr.	200.0
NV	52.4000	mg/cu.m	8 hr.	42.0
VA	220.0000	ug/cu.m	24 hr.	100.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

Colorless gas [30].

Odor Effects:

Mild aromatic or gasoline-like odor [30].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.13 2-BUTANONE (78-93-3)

Cancer Effects:

Not classified as a human carcinogen [31].

Non-Cancer (Systemic) Effects:

The EPA has issued an oral RfD based on two supporting animal studies [32]. The oral RfD is 5×10^{-2} mg/kg/day (175 ug/cu.m)a.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 31000 ug/cu.m

MOE (Standard) 24 hr. avg. 31000 ug/cu.m

Prop. 308 1 hr. avg. 31000 ug/cu.m

Advisories:

MOL TWEAV 200 ppm (590 mg/cu.m)

MOL STEV 300 ppm (885 mg/cu.m)

ACGIH TLV-TWA 200 ppm (590 mg/cu.m)

ACGIH STEL 300 ppm (885 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	1800.0000	ug/cu.m	8 hr.	50.0
MA	160.0000	ug/cu.m	24 hr.	0.0
NC	500.0000	ug/cu.m	15 min.	10.0
NV	14.0480	mg/cu.m	8 hr.	42.0
NY	000.0000	ug/cu.m	1 yr.	0.0
SD	800.0000	ug/cu.m	8 hr.	50.0
VA	800.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

Colorless gas [31].

Odor Effects:

Odor threshold 31000 ug/cu.m basis for MOE standard.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.14 BUTYL ACRYLATE (141-32-2)

Cancer Effects:

Not classified as a human carcinogen [33].

Non-Cancer (Systemic) Effects:

Irritation of respiratory tract follows inhalation. Inhalation of large doses results in pulmonary edema. Prolonged skin contact results in damage to the skin. No reports of injury are known due to long-term inhalation [34].

Standards:

Air:

MOE (Guideline) 0.5 hr. avg. 100 ug/cu.m

MOE (Guideline) 24 hr. avg. 100 ug/cu.m

Prop. 308 24 hr. avg. 35 ug/cu.m

Advisories:

MOL TWEAV 10 ppm (52 mg/cu.m)

ACGIH TLV-TWA 10 ppm (55 mg/cu.m)

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Unpleasant, persistent sweet odor. Thresholds for n-butyl acrylate reported as quite noticable at 0.9 ppm (4.7 mg/cu.m) and faintly noticable at less than 0.12 ppm (0.6 mg/cu.m) [33].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.15 CADMIUM (7440-43-9)

Cancer Effects:

Classified as a probable human carcinogen by inhalation [35] and as a suspected human carcinogen by the ACGIH [36]. Basis of classification is limited evidence from epidemiologic studies and sufficient evidence of carcinogenicity in rats and mice by two routes [35]. MOE's Phase III Dossier, however, states that there is only limited evidence that Cd exposure is associated with cancer of the prostate and lung. This evidence was derived from occupationally exposed populations. Therefore, the Dossier argues that the potential cancer risks to the general population must be considered light [37]. Classified as a 2B carcinogen by IARC, B by MAK, b by NTP, and as a carcinogen by NIOSH [2]. Massachusetts unit risk-- $1.80\text{E}-3$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

The principal tissue acutely affected by inhalation exposure is the lung. Exposure to high levels results in severe bronchial and pulmonary irritation [38]. Following chronic low-level inhalation exposure, the tissues most affected are the lung and kidney. Respiratory effects are indicative of chronic obstructive lung disease, renal injury by microglobinuria. ATSDR MRLs for cadmium inhalation are: acute exposures-- 0.005 mg/cu.m chronic exposures-- 0.0005 mg/cu.m EPA Baltimore IEMP TVs: fetal/developmental-- 21.0 ug/cu.m liver-- 0.4 ug/cu.m respiratory-- 2.0 ug/cu.m kidney-- 0.24 ug/cu.m reproductive-- 119.0 ug/cu.m.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 5 ug/cu.m

MOE (Standard) 24 hr. avg. 2 ug/cu.m

Prop. 308 24 hr. avg. 2 ug/cu.m

Water:

MOE water quality objective is 0.2 ug/L in unfiltered sample to protect aquatic life. Ontario max. accept. conc. 0.005 mg/L Ontario water criterion for livestock is 0.05 mg/L Ontario recommended max. conc. in irrigation water is 0.010 mg/L for waters used continuously on all soils and 0.050 mg/L

Advisories:

MOL TWAEV 0.05 mg/cu.m
 MOL STEV 0.2 mg/cu.m
 NIOSH TWA lowest
 IDLH 40 mg/cu.m
 ACGIH TLV-TWA 0.01 mg/cu.m
 NAS Suggested No Adverse Effect Level:
 Chronic 0.005 mg/L
 7 day 0.021 mg/L
 1 day 0.150 mg/L

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	0.4000	ug/cu.m	8 hr.	100.0
MA	0.0056	ug/cu.m	24 hr.	0.0
MT	0.3900	ug/cu.m	24 hr.	128.0
	0.0700	ug/cu.m	annual	714.0
NC	0.2500	ug/cu.m	24 hr.	200.0
NV	0.0010	mg/cu.m	8 hr.	42.0
NY	2.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	0.1200	ug/cu.m	1 yr.	420.0
RI	0.0006	ug/cu.m	annual	0.0
SD	0.4000	ug/cu.m	8 hr.	100.0
VA	0.8000	ug/cu.m	24 hr.	60.0

Animal Effects:

LD50 for cadmium oxide following 10-30 min. exposures range from 500-4000 mg/cu.m Little data for chronic, low level inhalation exposures.

Phytotoxic Effects:

Cadmium is phytotoxic to crops and trees [37].

Aquatic Ecosystem Effects:

Generally, available data indicate that fresh and salt water species show adverse effects from lower levels of cadmium under conditions of prolonged exposure, indicating that time is required for accumulation of the metal and the development of adverse effects. Consequently, short-term toxicity assessments may be of little relevance with respect to understanding potential hazards to aquatic life due to cadmium [37]. See [37] for numerous references.

Surface Water Effects:

See standards.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.16 CALCIUM CYANIDE (592-01-8)

Cancer Effects:

Not considered a carcinogen.

Non-Cancer (Systemic) Effects:

The EPA developed a RfD for chronic oral exposure of 4×10^{-2} mg/kg/day (140 ug/cu.m)a. RfD was based on two studies that established a NOAEL of 10.8 mg/kg/day and a LOAEL of 30 mg/kg/day [39].

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 100 mg/cu.m
MOE (Prov. Guid.) 24 hr. avg. 100 ug/cu.m
Prop. 308 24 hr. avg. 35 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.17 CAPTAN (133-06-2)

Cancer Effects:

The carcinogenicity of captan is under review by the EPA [40]. According to McCloskey [41], the carcinogenicity of Captan is controversial, but evidence is sufficient to recommend that exposure to captan be kept at the lowest practical level. It is not considered carcinogenic by the ACGIH [42].

Non-Cancer (Systemic) Effects:

The acute and subacute toxicity of captan has been widely studied [41]. Captan is not acutely toxic and is well tolerated at relatively high doses. The EPA developed a RfD for chronic oral exposure of $1.3\text{E-}2$ mg/kg/day (45.5 ug/cu.m)a.

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 75 ug/cu.m

MOE (Prov. Guid.) 24 hr. avg. 25 ug/ cu.m

Prop. 308 24 hr. avg. 25 ug/cu.m

Advisories:

MOL TWEAV 5 mg/cu.m

ACGIH TLV-TWA 5 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	100.0000	ug/cu.m	8 hr.	50.0
KS	11.9050	ug/cu.m	annual	420.0
NV	0.1190	mg/cu.m	8 hr.	42.0
PA-Phil.	35.0000	ug/cu.m	1 yr.	10.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.18 CARBON TETRACHLORIDE (56-23-5)

Cancer Effects:

The EPA considers carbon tetrachloride to be a probable human carcinogen [43]. The basis of this finding is carcinogenicity in rats, mice, and hamsters. Human data report liver tumor development after exposure to carbon tetrachloride. The Phase III Dossier concluded that it is reasonable to regard carbon tetrachloride as if it presented a carcinogenic risk to humans [44]. Classified as a 2B carcinogen by IARC, B by MAK, b by NTP, A2 by ACGIH, and as a carcinogen by NIOSH [2]. Massachusetts unit risk-- $1.50E-5$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

Carbon tetrachloride causes depression of the central nervous system at higher concentrations; lower concentrations (10-35 ppm or 62-217 mg/cu.m) cause loss of consciousness, dizziness, vertigo, headache, depression, mental confusion, and incoordination. Acute and chronic exposure results in kidney and liver tissue damage that results in impaired function. The EPA developed a RfD for chronic oral exposure of $7E-4$ mg/kg/day (2.45 ug/cu.m) [43]. EPA Baltimore IEMP TVs: liver--2.45 ug/cu.m neurobehavioral--2.45 ug/cu.m kidney--108 ug/cu.m reproductive--430 ug/cu.m fetal/developmental--24.2 ug/cu.m.

Standards:

Air:

MOE 0.05 hr. avg. 1800 ug/cu.m
 MOE 24 hr. avg. 600 ug/cu.m
 Prop. 308 24 hr. avg. 600 ug/cu.m
 U.S. Clean Water Act
 U.S. Ambient Water Quality Criteria
 Water and Fish Consumption $4.0E-1$ ug/L
 Fish Consumption Only $6.94E+0$ ug/L
 Acute LEC $3.52E+4$ ug/L
 Chronic LEC None

Advisories:

MOL TWAEV 5 ppm (31 mg/cu.m) (skin)
 MOL STEV 126 mg/cu.m
 OSHA TLV 100
 ACGIH TLV-TWA 5 ppm (30 mg/cu.m)
 NIOSH TWA 12.6 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	300.0000	ug/cu.m	8 hr.	100.0
IN	0.0667	ug/cu.m	annual	0.0
MA	0.6700	ug/cu.m	24 hr.	0.0
NC	150.0000	ug/cu.m	24 hr.	200.0
NV	0.7140	mg/cu.m	8 hr.	42.0
NY	100.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	72.0000	ug/cu.m	1 yr.	420.0
RI	0.0300	ug/cu.m	annual	0.0
VA	300.0000	ug/cu.m	24 hr.	100.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

21-200 ppm (130-1240 mg/cu.m); distinct
odor at 250 ppm (1550 mg/cu.m)
Odor threshold 21.4 ppm (133 mg/cu.m),
4.35 mg/L [9].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.19 CHLORDANE (57-74-9)

Cancer Effects:

The EPA considers chlordane to be a probable human carcinogen based on studies in which liver tumors were induced in mice and rats [45]. It is not considered carcinogenic by the ACGIH [46]. Classified as a B carcinogen by MAK [2]. IRIS inhalation slope factor-- $1.3E+0$ mg/kg/day IRIS inhalation unit risk-- $3.7E-4$ ug/cu.m.

Non-Cancer (Systemic) Effects:

In acute toxicity studies chlordane is moderately toxic to experimental animals. Animal studies showed chlordane had no significant effect on reproduction and metabolism. The EPA developed a RfD for chronic oral exposure of $5E-5$ mg/kg/day (0.175 ug/cu.m) based on a chronic rat dietary study. A fatal oral dose to humans is between 6 and 60 grams.

Standards:

MOE (Guideline) 0.5 hr. avg. 15 ug/cu.m
 MOE (Guideline) 24 hr. avg. 5 ug/cu.m
 Prop. 308 24 hr. avg. 5 ug/cu.m

Advisories:

MOL TWEAV 0.5 mg/cu.m (skin)
 MOL STEV 2 mg/cu.m (skin)
 ACGIH TLV-TWA $.5$ mg/cu.m
 OSHA PEL 500 ug/cu.m
 IDLH 500 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	2.5000	ug/cu.m	8 hr.	200.0
KS	1.1900	ug/cu.m	annual	420.0
MA	0.0680	ug/cu.m	24 hr.	0.0
NV	0.0120	mg/cu.m	8 hr.	42.0
NY	1.7000	ug/cu.m	1 yr.	0.0
PA-Phil.	0.3500	ug/cu.m	1 yr.	10.0
VA	8.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.20 CHLORINE (7782-50-5)

Cancer Effects:

There is no evidence to indicate that chlorine is a human carcinogen [47].

Non-Cancer (Systemic) Effects:

Chlorine exposures of 0.02 ppm (0.6 mg/cu.m) for 4 to 20 minutes result in irritation; at 1.0 ppm (3 mg/cu.m), symptoms include burning of conjunctiva and scratchiness of the throat. Concentrations between 21 and 40 ppm (63 and 120 mg/cu.m) over 1/2 hour to 1 hour may cause pulmonary edema and congestion [47].

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 300 ug/cu.m
 MOE (Standard) 24 hr. avg. 150 ug/cu.m
 Prop. 308 24 hr. avg. 150 ug/cu.m

Advisories:

MOL TWEAV 1 ppm (3 mg/cu.m)
 MOL STEV 3 ppm (9 mg/cu.m)
 ACGIH TLV-TWA 0.5 ppm (1.45 mg/cu.m)
 ACGIH TLV-STEL 1 ppm (3 mg/cu.m)
 OSHA PEL C 3 mg/cu.m
 EEGL/SPEGL 3 ppm
 IDLH 30 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	60.0000	ug/cu.m	8 hr.	50.0
KS	7.1430	ug/cu.m	annual	420.0
MA	39.0000	ug/cu.m	24 hr.	0.0
NV	0.0710	mg/cu.m	8 hr.	42.0
NY	10.0000	ug/cu.m	1 yr.	0.0
VA	50.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

Chlorine is a known phytotoxicant; injury threshold for sensitive species between 150 ug/cu.m and 800 ug/cu.m for acute exposures [48].

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

Greenish yellow gas

Odor Effects:

Accepted odor detection threshold between 0.2 and 0.4 ppm (0.6 and 1.2 mg/cu.m) [47]. Has a pungent odor. Odor threshold 0.314 ppm (0.94 mg/cu.m) [9].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.21 CHLORINE DIOXIDE (10049-04-4)

Cancer Effects:

Not considered a carcinogen by the ACGIH [49].

Non-Cancer (Systemic) Effects:

Chlorine dioxide has been linked to respiratory irritation at concentrations of about 1 ppm (3 mg/cu.m). Concentrations of 5 ppm (15 mg/cu.m) are definitely irritating. Protracted exposure caused bronchitis and pronounced emphysema in a chemist exposed to chlorine dioxide for several years [49].

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 30 ug/cu.m
MOE (Standard) 24 hr. avg. 85 ug/cu.m
Prop. 308 24 hr. avg. 30 ug/cu.m

Advisories:

MOL TWA EV 0.1 ppm (0.3 mg/cu.m)
MOL STEV 0.3 ppm (0.9 mg/cu.m)
ACGIH TLV-TWA 0.1 ppm (0.3 mg/cu.m)
ACGIH STEL 0.3 ppm (0.9 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	6.0000	ug/cu.m	8 hr.	50.0
NV	0.0070	mg/cu.m	8 hr.	42.0
NY	1.0000	ug/cu.m	1 yr.	0.0
VA	5.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 0.1 ppm (0.3 mg/cu.m) [49].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.22 CHLOROFORM (67-66-3)

Cancer Effects:

Data regarding the carcinogenicity of inhaled chloroform in humans and animals are not currently available. However, studies in animals indicate that chloroform is a carcinogen by the oral route. The EPA has not calculated carcinogenicity risk factors for a lifetime exposure. Classified as a 2B carcinogen by IARC, B by MAK, b by NTP, A2 by ACGIH, and as a carcinogen by NIOSH [2]. Massachusetts unit risk-- $2.35E-5$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

Target organs for chloroform toxicity are the liver, kidney, and central nervous system. The liver and kidney are the most sensitive targets for systemic toxicity. High levels of toxic hepatitis and liver enlargement were found in workers exposed to chloroform at levels as low as 2 ppm (9.8 mg/cu.m) [50]. The EPA developed a RfD for chronic oral exposure of $1E-2$ mg/kg/day (35 ug/cu.m) based on a LOAEL of 12.9 mg/kg/day. ATSDR MRLs for chloroform are: acute exposures--0.14 ppm (0.686 mg/cu.m) intermediate exposures--0.01 ppm (0.49 mg/cu.m) EPA Baltimore IEMP TVs: fetal/developmental--2.4 ug/cu.m liver--35 ug/cu.m neurobehavioral--11.7 ug/cu.m kidney--22.5 ug/cu.m reproductive--2.4 ug/cu.m

Standards:

Air:

MOE (Guideline) 0.5 hr. avg. 1500 ug/cu.m

MOE (Guideline) 24 hr. avg. 500 ug/cu.m

Prop. 308 24 hr. avg. 500 ug/cu.m

Water:

U.S. Safe Drinking Water Act

Maximum Contaminant Level 0.10 mg/L

U.S. Clean Water Act

U.S. Ambient Water Quality Criteria

Water and Fish Consumption $1.9E-1$ ug/L

Fish Consumption Only $1.57E+1$ ug/L

Acute LEC $2.89E+4$ ug/L

Chronic LEC $1.24E+3$ ug/L

Advisories:

MOL TWEAV 10 ppm (49 mg/cu.m)

NIOSH TWA 9.78 mg/cu.m

ACGIH TLV-TWA 10 ppm (50 mg/cu.m)

OSHA PEL 50 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	250.0000	ug/cu.m	8 hr.	200.0
IN	1200.0000	ug/cu.m	8 hr.	200.0
MA	0.4300	ug/cu.m	24 hr.	0.0
NC	0.4300	ug/cu.m	annual	0.0
NV	1.1900	mg/cu.m	8 hr.	42.0
NY	167.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	120.0000	ug/cu.m	1 yr.	420.0
RI	0.0400	ug/cu.m	annual	0.0
VA	500.0000	ug/cu.m	24 hr.	100.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

Chloroform has caused reduced gemination of corn and wheat.

Aquatic Ecosystem Effects:

Freshwater fish LC50 range from 18 mg/L for rainbow trout to 300 mg/L for guppy.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

0.3 ppm (1.5 mg/cu.m) [51].
Odor threshold 3.3 mg/L [52].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.23 CHROMIUM (7440-47-3)

Cancer Effects:

The EPA considers Chromium VI to be a human carcinogen by the inhalation route based on a number of epidemiologic studies that are consistent across investigators and locations. Dose-response relationships for lung tumors have been established. Chromium (VI) IRIS inhalation slope factor-- $4.1E+1$ mg/kg/day Chromium (VI) IRIS inhalation unit risk-- $1.2E-2$ ug/cu.m Hexavalent chromium compounds have not produced lung tumors in animals by inhalation. Trivalent chromium compounds have not been reported as carcinogenic by any route [53]. Chromium (+hexavalent compounds) classified as a 1 carcinogen by IARC, a by NTP, A1 by ACGIH, and as a carcinogen by NIOSH [2]. Chromium carbonyl classified as a B carcinogen by MAK [2]. Chromium III and chromate VI classified as A2 carcinogens by MAK [2].

Non-Cancer (Systemic) Effects:

Chromium VI is irritating, and short-term high level exposure can result in adverse effects at the site of contact, such as skin ulcers, irritation and perforation of nasal mucosa, and irritation of the gastrointestinal tract. Chromium VI may also cause adverse effects in the kidney and liver. The respiratory tract is the target of intermediate and chronic inhalation exposure to Chromium VI [54]. ATSDR MRLs for Chromium VI inhalation are: intermediate exposure-- 0.00005 mg/cu.m chronic exposure-- 0.00005 mg/cu.m The EPA developed a RfD for chronic oral exposure to Chromium VI of $5E-3$ mg/kg/day (17.5 ug/cu.m)a. The EPA developed a RfD for chronic oral exposure to Chromium III of $1E+0$ mg/kg/day (3500 ug/cu.m)a. EPA Baltimore IEMP TVs: fetal/developmental-- 17.0 ug/cu.m liver-- 17.0 ug/cu.m reproductive-- 17.0 ug/cu.m nonspecific cell.-- 17.5 ug/cu.m.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 10 ug/cu.m

MOE (Standard) 24 hr. avg. 30 ug/cu.m

Prop. 308 24 hr. avg. 1.5 ug/cu.m

U.S. Clean Water Act

U.S. Ambient Water Quality Criteria

Water and Fish Consumption $1.7E+5$ ug/L

Fish Consumption Only $3.433E+6$ ug/L

Acute LEC $9.8E+2$ ug/L

Chronic LEC $1.2E+2$ ug/L

Advisories:

MOL TWEAV 0.5 mg/cu.m for Cr, Cr II,
and Cr III
MOL TWEAV 0.05 mg/cu.m for Cr IV
and Cr VI
ACGIH TLV-TWA 0.5 mg/cu.m for Cr,
Cr II, and Cr III
ACGIH TLV-TWA 0.05 mg/cu.m Cr IV,
Cr VI, and Chromite Ore Processing
NIOSH TLV 25 ug/cu.m
NIOSH TLV 1 ug/cu.m for Cr IV

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	2.5000	ug/cu.m	8 hr.	200.0
MA	0.0680	ug/cu.m	24 hr.	0.0
MT	0.3900	ug/cu.m	24 hr.	128.0
	0.0700	ug/cu.m	annual	714.0
NC	0.2500	ug/cu.m	24 hr.	200.0
NV	0.0120	mg/cu.m	8 hr.	42.0
NY	0.1670	ug/cu.m	1 yr.	0.0
PA-Phil.	0.1200	ug/cu.m	1 yr.	420.0
RI	0.0900	ng/cu.m	annual	0.0
VA	0.5000	ug/cu.m	24 hr.	100.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

Considered toxic to plants. Interferes with root uptake of nutrients and their transfer to plant tops. Cr VI more toxic than Cr III. Concentrations of Cr usually present in air are too low to have any significant effect on the growth or yield of vegetation [55].

Aquatic Ecosystem Effects:

Available data indicate chronic toxicity to freshwater aquatic life occurs at concentrations as low as 44 mg/L, possibly lower for species not tested [55].

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 2.5 ug/cu.m.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.24 COAL TAR PITCH VOLATILES (8007-45-2)

Cancer Effects:

The benzene soluble fraction of coal tar pitch volatiles is recognized as a known human carcinogen by the ACGIH and the MAK [56]. Classified as an A1 carcinogen by MAK, and A1 by ACGIH [2]. The EPA has not developed unit risk estimates for coal tar pitch volatiles.

Non-Cancer (Systemic) Effects:

Health effects due to coal tar pitch volatiles are primarily cancers of the lung and kidney.

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 3 ug/cu.m
MOE (Prov. Guid.) 24 hr. avg. 1 ug/cu.m
MOE (Prov. Guid.) 1 yr. avg. 0.02 ug/cu.m
Prop. 308 24 hr. avg. 1 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	2.0000	ug/cu.m	8 hr.	200.0
KS	0.0161	ug/cu.m	annual	0.0
NC	1.0000	ug/cu.m	24 hr.	200.0
NV	0.0050	mg/cu.m	8 hr.	42.0
PA-Phil.	0.4800	ug/cu.m	1 yr.	420.0
VA	2.0000	ug/cu.m	24 hr.	100.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.25 EPICHLOROHYDRIN (106-89-8)

Cancer Effects:

The EPA considers epichlorohydrin to be a probable human carcinogen based on multiple studies in rats and mice [57]. IRIS inhalation unit risk-- $1.2\text{E-}6$ ug/cu.m Human data are inadequate to establish a relationship between epichlorohydrin and cancer. Classified as a 2B carcinogen by IARC, and A2 by MAK [2]. It is not considered carcinogenic by the ACGIH [58].

Non-Cancer (Systemic) Effects:

Epichlorohydrin causes burning of eyes and nasal passages at 20 ppm (76 mg/cu.m), severe chemical burns on skin contact and a possible skin dermatitis [59]. The EPA developed a RfD for chronic exposure to epichlorohydrin of $2\text{E-}3$ mg/kg/day (7 ug/cu.m)a. Primary target organ for systemic effects is the kidney. Epichlorohydrin is irritating and systemically toxic by oral, percutaneous, and respiratory routes in animals [58].

Standards:

Unable to locate MOE air quality guideline or regulation.

Advisories:

MOL TWEAV 2 ppm (7.6 mg/cu.m) (skin)
 ACGIH TLV-TWA 2 ppm (8 mg/cu.m)
 OSHA PEL 5 ppm
 IDLH 100 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	20.0000	ug/cu.m	8 hr.	100.0
IN	50.0000	ug/cu.m	8 hr.	200.0
MA	2.7000	ug/cu.m	24 hr.	0.0
NC	8.3000	ug/cu.m	annual	0.0
NV	0.2380	mg/cu.m	8 hr.	42.0
NY	33.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	12.0000	ug/cu.m	1 yr.	4200.0
RI	200.0000	ug/cu.m	24 hr.	0.0
VA	160.0000	ug/cu.m	24 hr.	6.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 10 ppm (38 mg/cu.m) [60].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.26 ETHYL BENZENE (100-41-4)

Cancer Effects:

Ethyl benzene is not expected to be carcinogenic [61], [62].

Non-Cancer (Systemic) Effects:

Ethyl benzene is an irritant to the skin and mucous membranes. Ethyl benzene is irritating to eyes at about 200 ppm (870 mg/cu.m) [63]. Above 100 ppm (435 mg/cu.m) it is reported to cause fatigue, headache, and mild irritation. At higher concentrations, ethyl benzene has caused increased weights in the livers and kidneys of experimental animals [63]. Some data indicate that exposures in the range of 50 mg/cu.m may produce central nervous system effects. The EPA developed an oral RfD for chronic ethyl benzene exposure of 1E-1 mg/kg/day (350 ug/cu.m)a [64].

Standards:

MOE (Standard) 0.5 hr. avg. 4000 ug/cu.m
 MOE (Standard) 1 hr. avg. 4000 ug/cu.m
 Prop. 308 1 hr. avg. 4000 ug/cu.m
 U.S. Clean Water Act
 U.S. Ambient Water Quality Criteria
 Water and Fish Consumption 1.4 mg/L
 Fish Consumption Only 3.28 mg/L
 Acute LEC 32000 ug/L
 Chronic LEC None

Advisories:

MOL TWA EV 100 ppm (435 mg/cu.m)
 MOL STEV 125 ppm (540 mg/cu.m)
 ACGIH TLV-TWA 100 ppm (435 mg/cu.m)
 ACGIH STEL 125 ppm (545 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	8700.0000	ug/cu.m	8 hr.	50.0
MA	120.0000	ug/cu.m	24 hr.	0.0
NV	10.3750	mg/cu.m	8 hr.	42.0
NY	1450.0000	ug/cu.m	1 yr.	0.0
VA	7250.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 140 ppm (609 mg/cu.m) 2-2.6 mg/cu.m [65].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.27 ETHYLENE (74-85-1)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

No IRIS, ACGIH, EPA or MOE information available.

Standards:

Air:

MOE (Ten. Des. Std.) 0.5 hr. avg. 160 ug/cu.m

MOE (Ten. Des. Std.) 24 hr. avg. 40 ug/cu.m

Prop. 308 24 hr. avg. 40 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
VA	3.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

MOE Standard based on phytotoxicity of ethylene. A 1976 report by Linzon listed dose-effects of ethylene on different vegetation types and a plant sensitivity ranking. Harper [75] suggests threshold levels ranging from 0.002 ppm (2.4 ug/cu.m) to 0.12 ppm (145.4 ug/cu.m) for 24 hour exposures.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.28 ETHYLENE DIBROMIDE (106-93-4)

Cancer Effects:

NIOSH recommended that ethylene dibromide be treated as a potential carcinogen in the workplace. NIOSH concluded that ethylene dibromide is a carcinogen in rats and mice [66]. Classified as a 2B carcinogen by IARC, A2 by MAK, b by NTP, and A2 by ACGIH [2].

Non-Cancer (Systemic) Effects:

Direct contact with ethylene dibromide causes irritation and injury to skin and eyes. Exposure to vapor causes development of respiratory tract inflammation along with anorexia and headache. Long-term exposure to ethylene dibromide may cause injury to the lungs, liver, or kidney. EPA Baltimore IEMP TVs: reproductive male--1.75 ug/cu.m reproductive female--11.9 ug/cu.m liver--12.8 ug/cu.m kidney--12.8 ug/cu.m.

Standards:

Unable to locate MOE air quality guideline or regulation.

Advisories:

OSHA PEL 20 ppm
OSHA PEL C 30 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	755.0000	ug/cu.m	8 hr.	200.0
IN	720.0000	ug/cu.m	8 hr.	200.0
NC	0.0450	ug/cu.m	annual	0.0
PA-Phil.	2.4700	ug/cu.m	1 yr.	4200.0
VA	500.0000	ug/cu.m	24 hr.	100.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

Freshwater fish LC50 range from 2.8 mg/L carp to 160 mg/L loach.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 10-25 ppm.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.29 ETHYLENE DICHLORIDE (107-06-2)

Cancer Effects:

No evidence for the carcinogenicity of ethylene dichloride in humans has been reported [70]. NIOSH determined that ethylene dichloride should be controlled as an occupational carcinogen and is a carcinogen in mice and rats [71]. The EPA considers ethylene dichloride to be a probable human carcinogen based on induction of several tumor types in rats and mice [72]. Classified as a 3 carcinogen by IARC, B by MAK, and b by NTP [2]. IRIS inhalation slope factor-- $9.1E-2$ mg/kg/day IRIS inhalation unit risk-- $2.6E-5$ ug/cu.m.

Non-Cancer (Systemic) Effects:

Acute inhalation studies indicate irritation of mucous membranes, central nervous system depression, and congestion and degenerative effects on the liver, kidney, spleen, lungs, and adrenals. Irritation of mucous membranes has been noted in humans at doses above 60 ppm (240 mg/cu.m). EPA Baltimore IEMP TVs: liver--26 ug/cu.m neurobehavioral--26 ug/cu.m kidney--26 ug/cu.m gastrointestinal--26 ug/cu.m.

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 1200 ug/cu.m

MOE (Prov. Guid.) 24 hr. avg. 400 ug/cu.m

Prop. 308 24 hr. avg. 400 ug/cu.m

U.S. Clean Water Act

U.S. Ambient Water Quality Criteria

Water and Fish Consumption $9.4 E-1$ ug/L

Fish Consumption Only $2.43E+2$ ug/L

Acute LEC $1.8E+4$ ug/L

Chronic LEC $9.4E+3$ ug/L

Advisories:

MOL TWEAV 10 ppm (40 mg/cu.m)

ACGIH TLV-TWA 40 mg/cu.m

ACGIH-STEL 60 mg/cu.m

OSHA PEL 202 mg/cu.m

OSHA PEL C 405 mg/cu.m

NIOSH TLV 4 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	20.0000	ug/cu.m	8 hr.	50.0
IN	1000.0000	ug/cu.m	8 hr.	200.0
MA	0.3900	ug/cu.m	24 hr.	0.0
NC	0.0380	ug/cu.m	annual	0.0
NV	0.9520	mg/cu.m	8 hr.	42.0
NY	0.2000	ug/cu.m	1 yr.	0.0
PA-Phil.	148.0000	ug/cu.m	1 yr.	1000.0
RI	0.0400	ug/cu.m	annual	0.0
VA	650.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 6-10 ppm (24-40 mg/cu.m).

Odor threshold 4.3E-3 mg/L [73].

Odor threshold 2.5E-2 mg/L [74].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.30 ETHYLENE GLYCOL BUTYL ETHER (BUTYL CELLOSOLVE) (107-21-1)

Cancer Effects:

Not considered to be carcinogenic.

Non-Cancer (Systemic) Effects:

The EPA developed a RfD for chronic oral exposure to ethylene glycol of 2E+0 mg/kg/day (7000 ug/cu.m)a based on a study indicating increased mortality neutrophil count, water intake, kidney hemoglobin and hematocrit and chronic nephritis in rats [67].

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 350 ug/cu.m
MOE (Prov. Guid.) 24 hr. avg. 2400 ug/cu.m
Prop. 308 1 hr. avg. 300 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
MA	170.0000	ug/cu.m	24 hr.	0.0
NV	2.9760	mg/cu.m	8 hr.	42.0
VA	1000.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

528 ug/cu.m

50% recognition 0.35 ppm [68]

Materials Damage:

No effects expected due to present ambient air concentrations.

C.31 ETHYLENE GLYCOL BUTYL ETHER ACETATE (BUTYL CELLOSOLVE ACETATE)

Cancer Effects:

No IRIS, ACGIH, EPA or MOE information available.

Non-Cancer (Systemic) Effects:

No IRIS, ACGIH, EPA or MOE information available.

Standards:

Prop. 308 1 hr. avg. 425 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 528 ug/cu.m

Materials Damage:

No effects expected due to present ambient air concentrations.

C.32 ETHYLENE GLYCOL ETHYL ETHER (CELLOSOLVE)

Cancer Effects:

No IRIS, ACGIH, EPA or MOE information available.

Non-Cancer (Systemic) Effects:

Principal effects include teratogenicity, fetotoxicity, genotoxicity, and metabolic alterations. 10 ppm appears to be a clear NOAEL in rats and rabbits.

Standards:

Prop. 308 1 hr. avg. 665 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.33 ETHYLENE GLYCOL ETHYL ETHER ACETATE (CELLOSOLVE ACETATE)

Cancer Effects:

No IRIS, ACGIH, EPA or MOE information available.

Non-Cancer (Systemic) Effects:

No studies documenting adverse human health effects due to EGEEA have been reported [69]. However, inhalation teratology studies in rats and rabbits suggest that EGEEA possesses the teratogenicity and fetotoxicity of EGEE.

Standards:

Prop. 308 1 hr. avg. 180 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.34 ETHYLENE OXIDE (75-21-08)

Cancer Effects:

Ethylene oxide is a possible human carcinogen. It is a known carcinogen in several rodent species. A suspected association with leukemia in humans cannot be confirmed or disconfirmed [76]. Classified as a 2B carcinogen by IARC, A2 by MAK, and A2 by ACGIH [2].

Non-Cancer (Systemic) Effects:

Acute exposures to ethylene oxide cause acute illness with nausea, vomiting, and headache. Animals exposed to ethylene oxide levels between 100 and 400 ppm (180 and 720 mg/cu.m) for periods up to 6 months have demonstrated growth depression, and neurological, renal, testicular, and hematological toxicity [77]. Neurological effects have been reported above and below 50 ppm (90 mg/cu.m), and various genotoxic effects have been observed in the workplace below 10 ppm (18 mg/cu.m).

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 15 ug/cu.m
MOE (Prov. Guid.) 24 hr. avg. 5 ug/cu.m
Prop. 308 24 hr. avg. 5 ug/cu.m

Advisories:

MOL TWEAV 1 ppm (1.8 mg/cu.m)
MOL STEV 10 ppm (18 mg/cu.m)
ACGIH TLV-TWA 1ppm (2 mg/cu.m)
OSHA PEL 1 ppm
EEGL/SPEGL 20 ppm
IDLH 800 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg.Time	Factor
CT	20.0000	ug/cu.m	8 hr.	100.0
IN	450.0000	ug/cu.m	8 hr.	200.0
NC	0.1000	ug/cu.m	annual	0.0
NV	0.0480	mg/cu.m	8 hr.	42.0
NY	6.7000	ug/cu.m	1 yr.	0.0
PA-Phil.	4.8700	ug/cu.m	1 yr.	4200.0
RI	0.0100	ug/cu.m	annual	0.0
SD	20.0000	ug/cu.m	8 hr.	100.0
VA	20.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 430-1500 ppm (774-2700 mg/cu.m).

50% recognition 500 ppm [78].

Odor detectable at about 700 ppm [79].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.35 FLUORIDES TOTAL (16984-48-8)

Cancer Effects:

Fluorides are not considered carcinogenic by the ACGIH [80].

Non-Cancer (Systemic) Effects:

Toxic effects of fluorides have been classified into 3 groups: acute systemic intoxication; corrosion of mucous membranes and skin; and chronic bone changes ranging from tooth enamel mottling to severe skeletal abnormalities.

Standards:

Air:

Prop. 308 30 day avg. 0.68 ug/cu.m, 24 hr. avg.

1.72 ug/cu.m (total, April 15 to Oct. 15)

Prop. 308 30 day avg. 1.38 ug/cu.m, 24 hr. avg.

3.44 ug/cu.m (total, Oct. 16 to April 14)

Gaseous and Particulate

April 1-September 30

AAQC 1.72 ug/cu.m 24 hrs.

AAQC 1.38 ug/cu.m 30 days

AAQC 0.5 hr. avg. 17.2 ug/cu.m

Advisories:

MOL TWEAV 2.5 mg/cu.m

ACGIH TLV-TWA 2.5 mg/cu.m, as F

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
IA	2.8500	ug/cu.m	24 hr.	0.0
KY	80.0000	ppm	monthly	0.0
MA	34.0000	ug/cu.m	24 hr.	0.0
MT	35.0000	ppm	graz.sea	0.0
NC	0.2500	mg/cu.m	1 hr.	0.0
VA	40.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

Standard set to protect animals from adverse effects due to elevated fluoride levels in forage. Recommended that fluoride

content of foliage should not exceed 60 ppm for more than 2 consecutive months, nor exceed 35 ppm dry weight of monthly results for growing season and not exceed 80 ppm dry weight for any single month.

Phytotoxic Effects:

For gaseous HF, injury threshold is approximately 1 ppb on gladiolus and potentially tulip [81].

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.36 FORMALDEHYDE (50-00-0)

Cancer Effects:

Formaldehyde produces squamous cell carcinomas in rats and mice in the nasal cavity at concentrations of 5.6 and 14.3 ppm by inhalation [82]. It doesn't induce tumors at other sites in mice and rats. Epidemiological studies have not demonstrated a relationship between formaldehyde exposure and nasal cancer [82]. Evidence suggests that formaldehyde caused tissue damage and, by implication, carcinogenesis, does not occur at exposure levels of less than approximately 1 ppm [82]. Classified as a 2B carcinogen by IARC, B by MAK, b by NTP, and A2 by ACGIH [2]. Massachusetts unit risk-- $1.30E-5$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

Non-lethal human effects are irritation of mucous membranes of eyes, nose and upper respiratory tract. Threshold is approximately 0.1 ppm (0.15 mg/cu.m). Discomfort begins at about 2-3 ppm (3-4.5 mg/cu.m). Symptoms include coughing, sneezing, lacrimation, feeling of suffocation, headache, increased pulse, fluctuations of body temperature and weakness. Exposure to concentrations greater than 5 ppm (7.5 mg/cu.m) can cause damage to the respiratory tract. Cases of formaldehyde asthma, respiratory tract irritation and dermatitis have been reported in exposed workers [83]. EPA Baltimore IEMP TV: nonspecific cellular effects--12.3 ug/cu.m.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 65 ug/cu.m

MOE (Standard) 1 hr. avg. 65 ug/cu.m

Prop. 308 1 hr. avg. 65 ug/cu.m

Advisories:

MOL TWEAV 1 ppm (1.5 mg/cu.m)

MOL STEV 2 ppm (3 mg/cu.m)

ACGIH TLV-TWA 1 ppm (1.5 mg/cu.m)

OSHA PEL 3 ppm

PEL C 5 ppm

IDLH 100 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	12.0000	ug/cu.m	8 hr.	100.0
IN	18.0000	ug/cu.m	8 hr.	200.0
KY	0.0000			0.0
MA	0.7700	ug/cu.m	24 hr.	0.0
NC	300.0000	ug/cu.m	15 min.	10.0
NV	0.0710	mg/cu.m	8 hr.	42.0
NY	2.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	7.2000	ug/cu.m	1 yr.	420.0
SD	12.0000	ug/cu.m	8 hr.	100.0
VA	12.0000	ug/cu.m	24 hr.	100.0
WA-Olympia	0.0500	ppm		0.5

Animal Effects:

Formaledehyde has been observed to cause excess mortalities in fish and amphibians, and to cause histological and cytological changes in the adrenal glands of pelicans [83].

Phytotoxic Effects:

Limited evidence suggests formaldehyde can depress plant photosynthesis under laboratory conditions [83].

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

Colorless gas [84].

Odor Effects:

Odor threshold 1 ppm (1.5 mg/cu.m) [85].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.37 HEXACHLOROCYCLOPENTADIENE (77-47-4)

Cancer Effects:

Not believed to be carcinogenic [86], [87].

Non-Cancer (Systemic) Effects:

Animal exposure to hexachlorocyclopentadiene vapor causes lacrimation, salivation, and gasping respiration and at high concentrations, tremors. Diffuse degenerative changes were observed in the brain, heart, liver, adrenal glands and kidneys [87]. In humans, inhalation of unknown quantities caused headaches in laboratory workers. Inhalation can cause pulmonary edema. Plant workers experienced skin and eye irritation, sore throat, cough, chest discomfort, headache, nausea and fatigue due to subacute-chronic exposures. Many workers had abnormal liver function. Inhalation exposures are unknown [83] EPA oral RfD is $7E-3$ mg/kg/day (24.5 ug/ cu.m)a [88].

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 6 ug/cu.m

MOE (Prov. Guid.) 24 hr. avg. 2 ug/cu.m

Prop. 308 24 hr. avg. 2 ug/cu.m

Advisories:

MOL TWEAV 0.01 ppm (0.11 mg/cu.m)

ACGIH TLV-TWA 0.01 ppm (0.1 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit.	Avg. Time	Factor
CT	2.0000	ug/cu.m	8 hr.	50.0
IN	0.5000	ug/cu.m	8 hr.	200.0
MA	0.0150	ug/cu.m	24 hr.	0.0
NC	0.0010	ppm	1 hr.	0.0
NV	0.0020	mg/cu.m	8 hr.	42.0
NY	0.3300	ug/cu.m	1 yr.	0.0
VA	2.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

Extremely toxic to fish and Daphnia in aqueous solution [86].

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 0.15-0.33 ppm

(1.7-3.6 mg/cu.m)

TOC 0.15 ppm (1.7 mg/cu.m) [89].

0.15 ppm (1.7 mg/cu.m) [87].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.38 HYDROGEN CHLORIDE (7647-01-0)

Cancer Effects:

Not believed to be carcinogenic [90].

Non-Cancer (Systemic) Effects:

Hydrogen chloride is a gas that readily dissolves in water producing hydrochloric acid. This is irritating to the eyes, nose, and throat. At high concentrations it can injure the lungs and ultimately cause death. Hydrogen chloride levels of 7,400 ug/cu.m are cited as causing irritation to mucous membranes [90]. Levels below this value are believed not to cause sub-acute or chronic effects.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 100 ug/cu.m

MOE (Standard) 24 hr. avg. 40 ug/cu.m

Prop. 308 24 hr. avg. 40 ug/cu.m

Advisories:

ACGIH-C 5 ppm (7mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
MA	10.0000	ug/cu.m	24 hr.	0.0
NV	0.1670	mg/cu.m	8 hr.	42.0
NY	140.0000	ug/cu.m	1 yr.	0.0
RI	2000.0000	ug/cu.m	1 hr.	0.0
SD	140.0000	ug/cu.m	8 hr.	50.0
VA	120.0000	ug/cu.m	24 hr.	0.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

MOE Standard may not provide adequate protection to vegetation [91].

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 1 ppm (1.4 mg/cu.m) [92]. Possesses a suffocating pungent odor [93].

Materials Damage:

Hydrogen chloride is a corrosive gas, MOE standards were set to protect materials from corrosion effects. Threshold at 40 ug/cu.m for 24 hours.

C.39 HYDROGEN CYANIDE (74-90-8)

Cancer Effects:

Not considered a carcinogen.

Non-Cancer (Systemic) Effects:

Hydrogen cyanide is extremely toxic and exposure often results in fatality. Symptoms include: dizziness, numbness, headache, rapid pulse, and blood-shot eyes. More prolonged exposure can cause vomiting, labored breathing, unconsciousness, and death [94]. Slight symptoms are experienced after exposure to 18-36 ppm (19.8-39.6 mg/cu.m) for several hours. It is immediately fatal at 270 ppm (297 mg/cu.m) [95]. EPA oral RfD is 2E-2 mg/kg/day (70 ug/cu.m)a [96].

Standards:

Prop. 308 24 hr. avg. 575 ug/cu.m

Advisories:

ACGIH-C 10 ppm (11 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	220.0000	ug/cu.m	8 hr.	50.0
NV	0.2380	mg/cu.m	8 hr.	42.0
NY	33.0000	ug/cu.m	1 yr.	0.0
VA	80.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

TOC 0.2-5 ppm (0.22-5.5 mg/cu.m) [97].

Odor threshold 1.0E-3 mg/L [98].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.40 HYDROGEN SULPHIDE (7783-06-4)

Cancer Effects:

Not considered carcinogenic by the ACGIH [99].

Non-Cancer (Systemic) Effects:

Exposure to very high concentrations (500-1000 ppm or 700-1400 mg/cu.m) causes death. Death or permanent injury may result after short exposures to small quantities. At low concentrations, contact with the eyes causes painful conjunctivitis, sensitivity to light and tearing. Other symptoms include salivation, nausea, vomiting, diarrhea, giddiness, dizziness, confusion, rapid breathing, weakness, sudden collapse, and unconsciousness [100]. The EPA oral RfD is 3E-3 mg/kg/day (10.5 ug/cu.m)a [100].

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 30 ug/cu.m

MOE (Standard) 1 hr. avg. 30 ug/cu.m

Prop. 308 1 hr. avg. 30 ug/cu.m

Advisories:

MOL TWEAV 10 ppm (14 mg/cu.m)

MOL STEV 15 ppm (21 mg/cu.m)

ACGIH TLV-TWA 10 ppm (14 mg/cu.m)

ACGIH TLV-STEL 15 ppm (21 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	280.0000	ug/cu.m	8 hr.	50.0
KY	0.0100	ppm	1 hr.	0.0
MA	19.0000	ug/cu.m	24 hr.	0.0
MT	50.0000	ppb	1 hr.	0.0
NV	0.3330	mg/cu.m	8 hr.	42.0
VA	230.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Standard based on odor threshold that ranges between 0.1-1100 ug/cu.m. Odor threshold 0.13 ppm (0.18 mg/cu.m) [98]. Odor threshold $1.8E-4$ mg/L [23]. Odor threshold $4.7E-3$ ppm ($6.6E-3$ mg/cu.m) [92]. Odor threshold $1.1E-3$ mg/L [101].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.41 INHALABLE PARTICULATES LESS THAN 10 UG [A]

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

See Suspended Particulate Matter for damage calculations.

Standards:

Unable to locate MOE air quality guideline or regulation.

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

See Suspended Particulate Matter.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.42 LEAD [A] (7439-92-1)

Cancer Effects:

Not considered carcinogenic by the ACGIH [102].

Non-Cancer (Systemic) Effects:

Health effects and welfare effects are documented in U.S. EPA's Criteria Document and RIA's. EPA Baltimore IEMP TVs: nonspecific cellular--1.5 ug/cu.m.

Standards:

Air:

MOE (Std. and Guid.) 3 ug/cu.m 30 d. mean
 MOE (Std. and Guid.) 2 ug/cu.m 30 d. geo. mean
 MOE (Std. and Guid.) 5 ug/cu.m sing. samp.
 0.5 hr. avg. 10 ug/cu.m
 Prop. 308 24 hr. avg. 5 ug/cu.m
 Prop. 308 30 day avg. 3 ug/cu.m (mean)
 Prop. 308 30 day avg. 2 ug/cu.m (goe. mean)
 U.S. 1.5 ug/cu.m calendar quarter avg.
 not to be exceeded; primary and secondary.

Advisories:

MOL TWEAV 0.15 mg/cu.m (0.10 mg/cu.m
 for tetraethyl lead)
 MOL STEV 0.30 mg/cu.m (0.45 mg/cu.m
 for tetraethyl lead)
 ACGIH TLV-TWA 0.15 mg/cu.m, as Pb

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	1.5000	ug/cu.m	8 hr.	100.0
IL	0.0000			0.0
KS	0.3570	ug/cu.m	annual	420.0
MA	0.0680	ug/cu.m	24 hr.	0.0
NV	0.0040	mg/cu.m	8 hr.	42.0
PA-Phil.	1.5000	ug/cu.m	1 yr.	1.0
VA	2.5000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.43 MANGANESE (7439-96-5)

Cancer Effects:

Insufficient data exist to class Mn as an animal carcinogen and no data on humans is available [103]. Not considered carcinogenic by the ACGIH [104].

Non-Cancer (Systemic) Effects:

Chronic manganese exposure results in damage to the central nervous system and the lungs. Reversible lung damage is believed to occur below 1 mg/cu.m in humans and pneumonia is associated with air concentrations between 0.1-14 mg/cu.m. Central nervous system effects usually occur above concentrations of 5 mg/cu.m. The WHO and the EPA suggest a health effect level between 1-10 ug/cu.m for chronic ambient exposure [103]. [103] recommends a chronic exposure health effects level of 250 ug/cu.m.

Standards:

Air:

MOE (Prov. Guid.) 24 hr. avg. 2.5 ug/cu.m

MOE (Prov Guid.) 0.5 hr. avg. 7.5 ug/cu.m

Prop. 308 24 hr. avg. 10 ug/cu.m

Advisories:

MOL TWEAV 1 mg/cu.m (fume)

MOL STEV 3 mg/cu.m (fume)

ACGIH-C 5 mg/cu.m.

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
	300.0000	ug/cu.m	15 min.	
NV	0.1190	mg/cu.m	8 hr.	42.0
PA-Phil.	25.0000	ug/cu.m	1 yr.	42.0
RI	2.0000	ug/cu.m	1 hr.	0.0
SD	20.0000	ug/cu.m	8 hr.	50.0
VA	17.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.44 MERCAPTANS

Cancer Effects:

No IRIS, ACGIH, EPA or MOE information available.

Non-Cancer (Systemic) Effects:

No IRIS, ACGIH, EPA or MOE information available.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 20 ug/cu.m

MOE (Standard) 1 hr. avg. 20 ug/cu.m

Prop. 308 1 hr. avg. 20 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Standard based on odor threshold.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.45 MERCURY (7439-97-6)

Cancer Effects:

No epidemiological studies have indicated that exposure to Mercury is associated with any form of cancer in humans [105]. Not considered carcinogenic by the ACGIH [106].

Non-Cancer (Systemic) Effects:

Acute exposures to high concentrations of mercury causes bronchial irritation, bronchitis, and diffuse interstitial pneumonitis [107]. Chronic exposure to mercury vapor results in anorexia, weight loss and minor central nervous system symptomatology, followed by mercurial tremor, psychic disturbances, and personality changes, gingivitis and stomatitis [107]. Long term exposure to levels of 0.1 mg/cu.m or greater may produce minimal changes in the neuro- psychiatric system of exposed workers.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 5 ug/cu.m

MOE (Standard) 24 hr. avg. 2 ug/cu.m

Prop. 308 24 hr. avg. 2 ug/cu.m

Advisories:

MOL TWEAV 0.05 mg/cu.m

MOL STEV 0.15 mg/cu.m

ACGIH TLV-TWA 0.05 mg/cu.m (Hg vapor)

ACGIH TLV-TWA 0.10 mg/cu.m (aryl and inorganic compounds)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	0.2000	ug/cu.m	8 hr.	50.0
IL	0.0000			0.0
KS	0.0240	ug/cu.m	annual	420.0
MT	0.0800	ug/cu.m	24 hr.	128.0
	0.0100	ug/cu.m	annual	714.0
NC	3.0000	ug/cu.m	15 min.	10.0
NV	0.0020	mg/cu.m		42.0
NY	0.1670	ug/cu.m	1 yr.	0.0
PA-Phil.	0.2400	ug/cu.m	1 yr.	42.0
VA	0.8000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Damage:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Effects:

No effects expected due to present ambient air concentrations.

C.46 MERCURY-ALKYL (7439-97-6)

Cancer Effects:

No epidemiological studies have indicated that exposure to Mercury is associated with any form of cancer in humans [105]. Not considered carcinogenic by the ACGIH [108].

Non-Cancer (Systemic) Effects:

The major target organ of alkyl mercury compounds is the brain. Neuropsychiatric effects of elemental and inorganic mercury are commonly reported. Earliest signs of damage due to alkyl mercury compounds are paresthesias and constriction of the visual field. In extreme cases coma and death occur. A threshold of 0.01 mg/cu.m has been proposed for alkyl-mercury airborne concentrations [107].

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 1.5 ug/cu.m
MOE (Standard) 24 hr. avg. 0.5 ug/cu.m
Prop. 308 24 hr. avg. 0.5 ug/cu.m

Advisories:

MOL TWEAV 0.01 mg/cu.m
MOL STEV 0.03 mg/cu.m
ACGIH TLV-TWA 0.10 mg/cu.m (alkyl compounds)
ACGIH STEL 0.03 mg/cu.m

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.47 METHANE DIPHENYL DI-ISOCYANATE

Cancer Effects:

No IRIS, ACGIH, EPA or MOE information available.

Non-Cancer (Systemic) Effects:

No IRIS, ACGIH, EPA or MOE information available.

Standards:

Air:

MOE (Tent. Des. Std.) 0.5 hr. avg. 3 ug/cu.m

MOE (Tent. Des. Std.) 24 hr. avg. 1 ug/cu.m

Prop. 308 24 hr. avg. 1 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient

Advisories:

MOL TWEAV 50 ppm (205 mg/cu.m)

MOL STEV 75 ppm (305 m/cu.m)

ACGIH TLV-TWA 50 ppm (205 mg/cu.m)

ACGIH TLV-STEL 75 ppm (300 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	0.0000			50.0
MA	280.0000	ug/cu.m	24 hr.	0.0
NC	30000.0000	ug/cu.m	15 min.	10.0
NV	1.1900	ppm	8 hr.	42.0
NY	680.0000	ug/cu.m	1 yr.	0.0
SD	4000.0000	ug/cu.m	8 hr.	50.0
VA	3400.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Effects:

No effects expected due to present ambient air concentrations.

C.48 METHYL ISOBUTYL KETONE (108-10-1)

Cancer Effects:

Not considered carcinogenic by the ACGIH [109].

Non-Cancer (Systemic) Effects:

High vapor concentrations irritate the conjunctive, mucous membranes of the nose and throat, producing eye and throat symptoms. Narcosis occurs with additional symptoms of weakness, headache, nausea, light headedness, vomiting, dizziness, and incoordination [109]. The EPA has issued an oral RfD based on one supporting animal study [110]. The oral RfD is $5E-2$ mg/kg/day (175 ug/cu.m).

Standards:

Air:

MOE (Guideline) 0.5 hr. avg. 1200 ug/cu.m

MOE (Guideline) 24 hr. avg. 1200 ug/cu.m

Prop. 308 24 hr. avg. 1200 ug/cu.m

Advisories:

MOL STEV 75 ppm (305 m/cu.m)

ACGIH TLV-TWA 50 ppm (205 mg/cu.m)

ACGIH TLV-STEL 75 ppm (300 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	0.0000			50.0
MA	280.0000	ug/cu.m	24 hr.	0.0
NC	30000.0000	ug/cu.m	15 min.	10.0
NV	1.1900	ppm	8 hr.	42.0
NY	680.0000	ug/cu.m	1 yr.	0.0
SD	4000.0000	ug/cu.m	8 hr.	50.0
VA	3400.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 1200 ug/cu.m.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.49 METHYLENE CHLORIDE (75-09-2)

Cancer Effects:

Classified as a probable human carcinogen by the EPA [111]. While the human data is considered inadequate, basis is increased cancer incidence in rats and mice. IRIS inhalation slope factor-- $1.4E-2$ mg/kg/day IRIS inhalation unit risk-- $4.1E-6$ ug/cu.m Classified as an A2 carcinogen by ACGIH [2].

Non-Cancer (Systemic) Effects:

Primary target organ is the central nervous system. Exposures above 500 ppm (1750 mg/cu.m) may cause sluggishness, irritability, light-headedness, nausea and headaches. Effects have been noted at 300 ppm (1050 mg/cu.m). Above 500 ppm (1750 mg/cu.m) methylene chloride can irritate the eyes, nose, and throat. The EPA has issued an oral RfD of $6E-2$ mg/kg/day (210 ug/cu.m) [111]. ATSDR [112] estimated MRLs for methylene chloride inhalation are: acute--5 ppm (17.5 mg/cu.m) intermediate--0.05 ppm (0.175 mg/cu.m) chronic--0.125 ppm (0.4375 mg/cu.m) EPA Baltimore IEMP TVs: liver--210.0 ug/cu.m fetal/developmental--210.0 ug/cu.m kidney--699.0 ug/cu.m neurobehavioral--8600.0 ug/cu.m.

Standards:

Air:

MOE (Ten. Des. Std.) 0.5 hr. avg. 5300 ug/cu.m

MOE (Ten. Des. Std.) 24 hr. avg. 1765 ug/cu.m

Prop. 308 1 hr. avg. 7000 ug/cu.

Water:

U.S. Clean Water Act

Ambient Water Quality Criteria

Water and Fish Consumption $1.9E-1$ ug/L

Fish Consumption Only $1.57E+1$ ug/L

Acute LEC $1.1E+4$ ug/L

Chronic LEC None

Advisories:

MOL TWEAV 50 ppm (175 mg/cu.m)

MOL STEV 500 ppm

ACGIH TLV-TWA 100 ppm (360 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	7000.0000	ug/cu.m	8 hr.	50.0
IN	8700.0000	ug/cu.m	8 hr.	200.0
KS	55.5500	ug/cu.m	annual	0.0
MA	2.4000	ug/cu.m	24 hr.	0.0
NC	1750.0000	ug/cu.m	24 hr.	200.0
NV	8.3330	mg/cu.m	8 hr.	42.0
NY	1200.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	8400.0000	ug/cu.m	1 yr.	42.0
RI	0.2000	ug/cu.m	annual	0.0
SD	3500.0000	ug/cu.m	8 hr.	100.0
VA	5800.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

96-hr. EC50 for both chlorophyll a inhibition and cell number decrease in *Selenasatrum capricornutum* 662 ppm [113].

Aquatic Ecosystem Effects:

Acute toxicity to freshwater life occurs at concentrations as low as 11 mg/L and may occur at lower concentrations among untested species [113].

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

100 ppm (350 mg/cu.m); 50% of population can identify at 214 ppm (750 mg/cu.m). Odor threshold 214 ppm (750 mg/cu.m). (92).

Materials Damage:

No effects expected due to present ambient air concentrations.

C.50 NAPHTHALENE (91-20-3)

Cancer Effects:

Not considered a carcinogen by the ACGIH [114].

Non-Cancer (Systemic) Effects:

Napthalene vapor may cause headache, loss of appetite, and nausea. Optical neuritis and injuries to the cornea, and kidney damage have also been reported [114]. The most significant health effects caused by napthalene are hemolytic anemia noted in humans following acute oral exposure, and effects on the respiratory tract (observed in mice) [115]. McCloskey derived a "safe" ambient exposure concentration of 22.5 ug/cu.m [115].

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 36 ug/cu.m
MOE (Prov. Guid.) 24 hr. avg. 22.5 ug/cu.m
MOE (Prov. Guid.) 10 min. avg. 50 ug/cu.m
Prop. 308 1 hr. avg. 30 ug/cu.m

Advisories:

MOL TWEAV 10 ppm (52 mg/cu.m)
MOL STEV 15 ppm (78 mg/cu.m)
ACGIH TLV-TWA 10 ppm (50 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	1000.0000	ug/cu.m	8 hr.	50.0
MA	14.0000	ug/cu.m	24 hr.	0.0
NV	1.1900	mg/cu.m	8 hr.	42.0
NY	166.7000	ug/cu.m	1 yr.	0.0
VA	800.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

50 ug/cu.m standard set to protect Odor threshold.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.51 NICKEL (7440-02-0)

Cancer Effects:

Nickel subsulfide is classified as a human carcinogen by the EPA [116] on the basis of increased risks of lung and nasal cancer in humans exposed to nickel refinery dust, most of which was believed to have been nickel subsulfide; increased tumor incidences in animals by several routes of exposure; and positive results in genotoxicity assays. Classified as a 3 carcinogen by IARC [2]. IRIS inhalation unit risk-- $4.8E-4$ ug/cu.m IRIS inhalation slope factor-- $1.7E+0$ mg/kg/day Nickel refinery dust is classified as a human carcinogen by the EPA [117] on the basis on human data in which exposure to nickel refinery dust caused tumors, and on animal data. Classified as a 1 carcinogen by IARC, A1 by MAK, a by NTP, and A1 by ACGIH [2]. IRIS inhalation slope factor-- $8.4E-1$ mg/kg/day IRIS inhalation unit risk-- $2.4E-4$ ug/cu.m.

Non-Cancer (Systemic) Effects:

The lung is the target organ of nickel toxicity in humans. Inhalation exposure to certain nickel compounds causes asthma and increased susceptibility to pulmonary infections. Chronic exposure to high levels may cause anosmia and severe nasal injury [118]. The EPA has issued an oral nickel, soluble salts, RfD of $2E-2$ mg/kg/day (70 ug/cu.m)a [119]. ATSDR [118] estimated MRLs for nickel compound inhalation are: intermediate-- 0.0009 mg/cu.m.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 5 ug/cu.m
 MOE (Standard) 24 hr. avg. 2 ug/cu.m
 Prop. 308 24 hr. avg. 2 ug/cu.m

Advisories:

MOL TWA EV 1 mg/cu.m
 Water Sol. Ni Compds. TWA EC 0.1 mg/cu.m
 Water Sol. Ni Compds. STEC 0.3 mg/cu.m
 ACGIH Ni dust TLV-TWA 1mg/cu.m
 ACGIH insol. compds. TLV-TWA 1.0 mg/cu.m
 ACGIH sol. compds. TLV-TWA 0.1 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
KS	0.0303	ug/cu.m	annual	0.0
MA	0.0180	ug/cu.m	24 hr.	0.0
MT	0.7900	ug/cu.m	24 hr.	128.0
	0.1300	ug/cu.m	annual	714.0
RI	0.0020	ug/cu.m	annual	0.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

Low level exposures result in growth inhibition with no visible symptoms. Chlorosis has been observed at high levels of exposure, including signs of light yellow and green banding [119].

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.52 NICKEL CARBONYL (13463-39-3)

Cancer Effects:

Classified as a probable human carcinogen by the EPA [120] on the basis of pulmonary carcinomas and malignant tumors at various sites in rats following inhalation and intravenous injection. EPA has not developed quantitative risk measures. Classified as an A2 carcinogen by MAK, and as a carcinogen by NIOSH [2]. Nickel carbonyl is not considered carcinogenic by the ACGIH [121].

Non-Cancer (Systemic) Effects:

Nickel carbonyl may cause headache, tightness in the chest, nausea, liver damage, and brain injury. The primary effect of acute exposure is to the lungs. 30 ppm (210 mg/cu.m) for 30 minutes may be lethal to humans [121].

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 1.5 ug/cu.m
MOE (Standard) 24 hr. avg. .5 ug/cu.m
Prop. 308 24 hr avg. 0.5 ug/cu.m

Advisories:

MOL TWEAV 0.05 ppm (0.35 mg/cu.m)
ACGIH TLV-TWA 0.05 ppm (0.35 mg/cu.m)
OSHA PEL 0.007 mg/cu.m
IDLH 0.0001 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	0.0350	ug/cu.m	8 hr.	200.0
NV	0.0080	mg/cu.m	8 hr.	42.0
NY	1.1700	ug/cu.m	1 yr.	0.0
VA	5.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Approximately 30 ppb (210 ug/cu.m) (121).

Materials Damage:

No effects expected due to present ambient air concentrations.

C.53 NITROGEN DIOXIDE [A] (10102-44-0)

Cancer Effects:

Not considered carcinogenic

Non-Cancer (Systemic) Effects:

EYE IRRITATION Y = change in eye irritation days per adult.
Lower Bound: Y = 1/2 Central Case Central Case: Y = 3.8724 *
NO₂DH%. Upper Bound: Y = 2 * Central Case. Where NO₂DH% = %
change in avg. daily high NO₂ in ppm. Values: Lower = Central:
\$5 1986 U.S.; Upper: \$12.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 500 ug/cu.m
MOE (Standard) 24 hr. avg. 200 ug/cu.m
Prop. 308 24 hr. avg. 200 ug/cu.m
U.S. Clean Air Act

Advisories:

MOL TWEAV 3 ppm (5.6 mg/cu.m)
MOL STEV 5 ppm (9.4 mg/cu.m)
ACGIH TLV-TWA 3 ppm (6 mg/cu.m)
ACGIH TLV-STEL 5 ppm (10 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
AZ-Pigico	100.0000	ug/cu.m	annual	0.0
CT	120.0000	ug/cu.m	8 hr.	50.0
NV	0.1430	mg/cu.m	8 hr.	42.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

Y = change in annual values per household per year \$1986 U.S.
Central Case: $Y = 205 * \ln(1/1 - (0.02 * PN_{02}))$ Rural. $Y = 205 * \ln(1/1 - (0.05 * PN_{02}))$ Urban. Lower Case: Replace 205 by 100. Replace 205 by 400. Where PN_{02} = % change in NO_2 annual average all hours in ppm.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

Y = change in annual values per person per year in \$1986 U. S.
Lower bound: $Y = 1/2 * \text{central Case}$. Central Case: $Y = 1.51 * PN_{02}$. Upper bound: $Y = 2 * \text{central case}$. Where PN_{02} = % change in NO_2 annual avg. all hours in ppm.

C.54 NITROUS OXIDE (10024-97-2)

Cancer Effects:

Not considered carcinogenic [122].

Non-Cancer (Systemic) Effects:

Chronic exposure to nitrous oxide has been linked to neuropathy, hemapoietic disorders, increases in spontaneous abortions, and suspicions of abnormalities [122]. The human reproductive, hematologic, and nervous systems appear to be most susceptible to nitrous oxide toxicity. The EPA has developed an oral nitrous oxide RfD of $1\text{E}-1$ mg/kg/day (350 ug/cu.m) based on an epidemiologic study of the incidence of methemoglobinemia in infants fed formula with nitrate-contaminated water [123].

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 27000 ug/cu.m

MOE (Prov. Guid.) 24 hr. avg. 9000 ug/cu.m

Prop. 308 24 hr. avg. 9000 ug/cu.m

Advisories:

MOL TWEAV 25 ppm (45 mg/cu.m)

ACGIH TLV-TWA 50 ppm (91 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg Time	Factor
CT	40.0000	ug/cu.m	8 hr.	50.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.55 OZONE [A] (10028-15-6)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

RESPIRATORY RESTRICTED ACTIVITY DAYS (RRADs)

Y = change in RRAD per person

1. First Set:

Lower Bound: $Y = \sum i (.71 * \text{change in OZ1})$

Central Case: Average of Upper and Lower

Upper Bound: $Y = \sum i (.254 * (\text{OZ1}^{**-.5}) * \text{change in OZ1})$

Where OZ1 = 2 week arithmetic avg. of daily high hour ozone as ppm, and i = 1 to 26

To use annual average:

Lower Case: $Y = 26 * .71 * (\text{change in annual avg.}) [\text{ppm}]$

Upper Case: $Y = 26 * .245 * (\text{change in annual avg.})^{**-.5}$

2. Second Set:

Lower Bound: $Y = .909 * \text{OZ\%}$

Central Case: $Y = 1.212 * \text{OZ\%}$

Upper Bound: $Y = 1.36 * \text{OZ\%}$

Where OZ% = % change in peak ozone readings.

ASTHMA ATTACKS

Assumes baseline # attacks/year/asthmatic = 50 and adult population = 70%. Y = change in attacks per asthmatic

Lower Bound: $Y = 0$ Best Case: $Y = 4.9 * \text{OZ\%} - \text{change in RRADs}$ Upper Bound: $Y = 7.7 * \text{OZ\%} - \text{change in RRADs}$

Where OZ% = % change in peak daily O3 (ppm). Values:

Upper: per case \$43 U.S. 1986; Central: \$26; Lower: \$9

EYE IRRITATION

Y = change in irritation days/year per adult Lower Bound:

$Y = 7.865 * \text{OZ\%}$ Central Case: $Y = 8.62 * \text{OZ\%}$ Upper Bound:

$Y = 9.35 * \text{OZ\%}$ Where OZ% = % change in annual peak O3

values (ppm). Values: Low = Central: \$5 U.S. 1986; High:

\$12 COUGH Y = change in cough days/year per adult Lower

Bound: Y = Central Case Central Case: $Y = 4.235 * \text{OZ\%} -$

change in RRADs Upper Bound: Y = Central Case Where OZ% = % change in annual peak O3 values (ppm). Value: Low =

Central: \$4 U.S. 1986; Upper: \$8 SUM OF MINOR SYMPTOM Y =

change in days with 1 or more minor symptoms for adults

Lower Bound: $Y = 3.63 * \text{OZ\%}$ Where OZ% = % change in daily peak O3 values (ppm).

Standards:

Air:

MOE (Standard) 0.5 hr. avg 200 ug/cu.m

MOE (Standard) 1 hr. avg. 165 ug/cu.m
(0.08 ppm)

Prop. 308 1 hr. avg. 165 ug/cu.m

Advisories:

MOL TWEAV 0.1 ppm (0.2 mg/cu.m)

MOL STEV 0.3 ppm (0.6 mg/cu.m)

ACGIH-C 0.1ppm (0.2 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	235.0000	ug/cu.m	1 hr	50.0
NV	0.0000	ug/cu.m	8 hr	42.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

Ornamentals $Y = \text{change in per capita damage } \1986 U.S. Central Case: $Y = 6.5 * \text{change in annual avg. O}_3 \text{ in ppm.}$ Known phytotoxicant, 165 ug/cu.m. threshold corresponds to 0.035 ppm. 3-month seasonal mean. This level is considered protective of crops and natural vegetation by the MOE [124].

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 1.0E-3 mg/L [125].

Materials Damage:

Y = change in annual per capita damage in \$1986 U.S. Lower
Bound: $Y = 8.68 * \text{change in O3AA}$ Central Case: $Y = 28.06 * \text{change in O3AA}$
Upper Case: $Y = 47.43 * \text{change in O3AA}$. Where:
O3AA = annual average ozone for all hours in ppm.

C.56 PENTACHLOROBENZENES (608-93-5)

Cancer Effects:

Not considered carcinogenic [126, 127].

Non-Cancer (Systemic) Effects:

Local irritation may result from eye and dermal contact. Irritation of the respiratory memberane may result from inhalation [126]. Ingestion of small amounts of pentachlorobenzene for protracted periods may result in cutaneous porphyria, hyperpigmentation, excess hair growth, liver enlargement, and deformities of digits. Central nervous system effects may include headache, dizziness, numbness and partial paralysis of extremities, and coma and convulsions. The EPA established an oral Rfd of $8E-4$ mg/kg/day (2.8 ug/cu.m)a [127].

Standards:

Unable to locate MOE air quality guideline or regulation.

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold $6.0E-2$ mg/L [126].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.57 PENTACHLOROPHENOL (87-86-5)

Cancer Effects:

Not considered a carcinogen by the ACGIH [128].

Non-Cancer (Systemic) Effects:

According to the ACGIH, the most important effect of pentachlorophenol inhalation is acute poisoning centering in the circulatory system with accompanying heart failure [128]. Pentachlorophenol is fatal at high oral doses [129]. Chronic exposure has resulted in conjunctivitis, chronic sinusitis, upper respiratory complaints, recurring headache, and neurological pains. The EPA has issued an oral pentachlorophenol RfD of $3\text{E-}2$ mg/kg/day (105 ug/cu.m) based on a chronic rat study that estimated a NOAEL of 3 mg/kg/day [130]. The same study was used by Argent to estimate an acceptable pentachlorophenol concentration in ambient air of 0.03 mg/cu.m [131].

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 90 ug/cu.m
MOE (Standard) 24 hr. avg. 30 ug/cu.m
MOE (Guideline) 0.5 hr. avg. 60 ug/cu.m
MOE (Guideline) 24 hr. avg. 20 ug/cu.m
Prop. 308 24 hr. avg. 20 ug/cu.m
U.S. Clean Water Act
Ambient Water Quality Criteria
Water and Fish Consumption $1.01\text{E+}3\text{ ug/L}$
Fish Consumption Only None
Acute LEC $1.3\text{E+}1\text{ ug/L}$
Chronic LEC $7.9\text{E+}0\text{ ug/L}$

Advisories:

MOL TWEAV 0.5 mg/cu.m (skin)
ACGIH TLV-TWA 0.5 mg/cu.m
OSHA PEL 0.5 mg/cu.m
IDLH 150 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	5.0000	ug/cu.m	8 hr.	100.0
KS	25.6400	ug/cu.m	annual	0.0
MA	0.0340	ug/cu.m	24 hr.	0.0
NC	2.5000	ug/cu.m	24 hr.	200.0
NV	0.0120	mg/cu.m	8 hr.	42.0
NY	1.6700	ug/cu.m	1 yr.	0.0
PA-Phil.	12.0000	ug/cu.m	1 yr.	42.0
SD	10.0000	ug/cu.m	8 hr.	100.0
VA	8.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

Rat NOAEL 3-10 mg/kg/day [132].

Phytotoxic Effects:

Effects range from increased yield to lethality, depending on dose and application [132].

Aquatic Ecosystem Effects:

Fathead minnow LOAEL 0.073 mg/L.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.58 PERCHLOROETHYLENE (127-18-4)

Cancer Effects:

Not considered a carcinogen by the ACGIH [133].

Non-Cancer (Systemic) Effects:

Excessive exposure to perchloroethylene has resulted in effects on the central nervous system, musous membranes, eyes, and skin, and to a lesser extent the Lungs, Liver and Kidneys (133). The EPA established an oral RfD of $1E-2$ mg/kg/day Liver--69.9 ug/cu.m Kidney--69.9 ug/cu.m Fetal/developmental 909.0 ug/cu.m

Standards:

Air:

MOE (Guideline) 0.5 hr. avg. 10000 ug/cu.m

Prop. 308 24 hr. avg. 4000 ug/cu.m

U.S.

Clean Water Act

Ambient Water Quality Criteria

Water and Fish Consumption $8.0E-1$ ug/L

Fish Consumption Only $8.85E+0$ ug/L

Acute LEC $5.28E+3$ ug/L

Chronic LEC $8.4E+2$ ug/L

Advisories:

MOL TWEAV 50 ppm (340 mg/cu.m)

MOL STEV 200 ppm (1355 mg/cu.m)

ACGIH TLV-TWA 50 ppm (340 mg/cu.m)

ACGIH TLV-STEL 200 ppm (1340 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
IN	3350.0000	ug/cu.m	8 hr.	200.0
KS	5.8820	ug/cu.m	annual	0.0
MA	0.1800	ug/cu.m	24 hr.	0.0
NC	134.0000	mg/cu.m	15 min.	10.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

TOC 50 ppm (340 mg/cu.m);

PIT50% 4.68 ppm (31.8 mg/cu.m) [135].

Odor threshold 4.68 ppm (31.8 mg/cu.m) [136].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.59 PHENOL (108-95-2)

Cancer Effects:

Does not appear to be carcinogenic [137], [138].

Non-Cancer (Systemic) Effects:

exposures resulting in death due to respiratory arrest, pulmonary edema, heart, or renal failure [139]. Chronic exposure can result in digestive disturbances, vomiting, diarrhea, nervous disorders [137]. Systemic effects have been noted in the liver, kidney, and central nervous system. Harding calculated the maximum daily allowable intake in ambient air as 0.3 mg/cu.m [139]. The EPA oral RfD is 4E-2 mg/kg/day (140 ug/cu.m)a [140]. EPA Baltimore IEMP TVs:
liver--40 ug/cu.m kidney--40 ug/cu.m.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 100 ug/cu.m

MOE (Standard) 24 hr. avg. 45 ug/cu.m

Prop. 308 24 hr. avg. 100 ug/cu.m

Water:

MOE Drinking Water Max. Lev. 0.002 mg/L

MOE Aquatic Life 1.0 ug/L

Advisories:

MOL TWEAC 5 ppm (19 mg/cu.m) (skin)

MOL STEL 10 ppm

ACGIH TLV-TWA 5 ppm (19 mg/cu.m)

OSHA PEL 19 mg/cu.m

IDLH 250 ppm

Ambient Air Levels:

State	Conc	Unit	Avg. Time	Factor
CT	380.0000	ug/cu.m	8 hr.	50.0
IN	95.0000	ug/cu.m	8hr.	200.0
KS	45.2380	ug/cu.m	annual	420.0
MA	52.0000	ug/cu.m	24 hr.	0.0
NC	95.0000	ug/cu.m	24 hr.	200.0
NV	0.4520	mg/cu.m	8 hr.	42.0
NY	10.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	456.0000	ug/cu.m	1 yr.	42.0
SD	380.0000	ug/cu.m	8 hr.	50.0
VA	315.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effect due to present ambient air concentrations.

Phytotoxic Effects:

Chronic plant toxicity Limit 20 ppm, irrigable plants 50 ppm [137].

Aquatic Ecosystem Effects:

Very toxic to aqualtic life. LC50's range from 0.39 mg/L for goldfish to 9 ug/L for rainbow trout to 36 mg/L for zebra- fish and 56 mg/L for mosquito fish [137]. Toxic to many species of macroinvertebrates.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effect expected due to present ambient air concentrations.

Odor Effects:

0.016-16.7 ppm (0.06-63.5 mg/cu.m)

Odor threshold 4.7E-2 ppm (0.18 mg/ cu.m) [141].

Odor threshold 1 ppm (3.8 mg/cu.m) [142].

Materials Damage:

Corrosive to aluminum and alloys, rubber, and lead.

C.60 PHOSGENE (75-44-5)

Cancer Effects:

Not considered a carcinogen by the AGGIH [143].

Non-Cancer (Systemic) Effects:

May cause irritation of throat and eyes, coughing, acute pulmonary edema, death, emphysema, and fibrosis. May cause chronic lung diseases in man. Data from chemical warfare service prior to 1921 indicated that 1 ppm (4 mg/cu.m) of phosgene may be considered safe for prolonged exposure [143].

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 130 ug/cu.m

MOE (Standard) 24 hr. avg. 45 ug/cu.m

Prop. 308 24 hr. avg. 45 ug/cu.m

Advisories:

ACGIH TLV-TWA 0.1 ppm (0.4 mg/cu.m)

OSHA PEL .4 mg/cu.m

EEGL/SPEGL .2 ppm

IDLH 2 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	0000	ug/cu.m	8 hr.	50.0
NC	2.0000	ug/cu.m	24 hr	200.0
NV	0.0100	mg/cu.m	8 hr.	42.0
NY	1.3300	ug/cu.m	1 yr.	0.0
VA	7.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Strong irritating smell, 2 ppm (8 mg/cu.m) [144]. Odor threshold 1 ppm (2 mg/cu.m) [145].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.61 PHOSPHINE (7803-51-2)

Cancer Effects:

No considered a carcinogen.

Non-Cancer (Systemic) Effects:

Phosphine is a very toxic gas with a probable oral lethal dose of 5 mg/kg. An air concentration of 3 ppm (4 mg/cu.m) is safe for long term exposure, 500 ppm (667 mg/cu.m) is lethal in 30 minutes [146]. The EPA developed an oral RfD of $3E-4$ mg/kg/day (1 ug/cu.m)a [146].

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 30 ug/cu.m

MOE (Standard) 24 hr. avg. 10 ug/cu.m

Prop. 308 24 hr. avg. 10 ug/cu.m

Advisories:

MOL TWEAV 0.3 ppm (0.4 mg.cu.m)

MOL STEV 1 ppm (1.4 mg/cu.m)

ACGIH TLV-TWA 0.3 ppm (0.4 mg/cu.m)

OSHA PEL .4 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	8.0000	ug/cu.m	8 hr	50.0
NC	100.0000	ug/cu.m	15hr	10.0
NV	0.0100	mg/cu.m	8 hr	42.0
NY	1.3300	ug/cu.m	1yr	0.0
VA	6.7000	ug/cu.m	24hr	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 2.1E ppm (0.028 mg/cu.m) [145] Disagreeable garlic like odor [147].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.62 POLYCHLORINATED BIPHENYLS (1336-36-3)

Cancer Effects:

Classified as a 2B carcinogen by IARC, by NTP, and as a carcinogen by NIOSH [2]. Massachusetts unit-risk-- $2.20\text{E}-3$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

Exposure to polychlorinated biphenyl vapor or fumes may cause acne, irritation of respiratory passage and injury to the liver. Air concentrations of 0.1 mg/cu.m caused mild to moderate chloracne. PCBs can be absorbed through the skin causing fatty degeneration of the liver [148].

Standards:

Air:

MOE (Guideline) 0.5 hr. avg. 450 ng/cu.m
MOE (Guideline) 1 yr. avg. 35 ng/cu.m
MOE (Guideline) 24 hr. avg. 150 ng/cu.m
Prop. 308 24 hr. avg. 150 ng/cu.m, 1 yr. avg.
35 ng/cu.m (risk: 0.84/million)

Advisories:

No workplace standards found.

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
KS	0.0083	ug/cu.m	annual	0.0
MA	0.0081	ug/cu.m	24	0.0
NY	1.6700	ug/cu.m	1 yr.	0.0
VA	8.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.63 POTASSIUM CYANIDE (151-50-8)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

Potassium cyanide is super toxic; probable oral lethal dose in humans is less than 5 mg/kg [149]. The EPA oral RfD is 5E-2 mg/kg/day (175 ug/cu.m)a [149].

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 100 ug/cu.m

MOE (Prov. Guid.) 24 hr. avg. 100 ug/cu.m

Prop. 308 24 hr. avg. 35 ug/cu.m

Advisories:

OSHA PEL 5 mg/cu.m

IDLH 50 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
NV	0.1190	mg/cu.m	8hr	42.0
NY	17.0000	ug/cu.m	1 yr	0.0
VA	80.0000	ug/cu.m	24 hr.	60.0

Aimal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.64 POTASSIUM HYDROXIDE (1310-58-3)

Cancer Effects:

Not considered carcinogenic by the ACGIH [150].

Non-Cancer (Systemic) Effects:

Potassium hydroxide gelatinizes tissue, forming soluble compounds that may result in deep and painful lesions [150].

Standards:

Air

MOE (Prov. Guid.) 0.5 hr. avg. 28 ug/cu.m

MOE (Prov. Guid.) 24 hr. avg. 14 ug/cu.m

Prop. 308 24 hr. avg. 14 ug/cu.m

Advisories:

ACHIH-C 2 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
NV	0.0480	mg/cu.m	8 hr	42.0
VA	16.0000	ug/cu.m	24 hr	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.65 PROPIONALDEHYDE (123-38-6)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

Insufficient data to set health base airborne exposure limit [151].

Standards:

Air:

Prop. 308 24 hr. avg. 2.5 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Detection Level 10 ug/cu.m Odor threshold $2.2\text{E-}3$ mg/L, $3.5\text{E-}7$ g/L [152].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.66 PROPIONIC ACID (79-09-4)

Cancer Effects:

No considered carcinogenic by the ACGIH [153].

Non-Cancer (Systemic) Effects:

Acute human exposures cause mild to moderate skin burns, mild eye response, and 1 case of mild cough and asthma [153].

Standards:

Air:

MOE (Guideline) 0.5 hr. avg. 100 ug/cu.m

MOE (Guideline) 24 hr. avg. 100 ug/cu.m

Prop. 308 1 hr. avg. 80 ug/cu.m

Advisories:

MOL TWEAV 10 ppm (30 mg/cu.m)

ACGIH TLV-TWA 10 ppm (30 mg/cu.m)

ACGIH TLV-STEL 15 ppm (45 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	600.0000	ug/cu.m	8 hr	50.0
NV	0.7140	mg/cu.m	8 hr	42.0
VA	500.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 2-900 ug/cu.m

Materials Damage:

No effects expected due to present ambient air concentrations.

C.67 PROPYLENE GLYCOL MONOMETHYL ETHER (107-98-2)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

PGME has relatively low toxicity via inhalation [154]. McCloskey notes mild central nervous system depression in animal exposures of 3000 ppm (10800 mg/cu.m) [154]. Human volunteers found PGME irritating above 100 ppm (360 mg/ cu.m) [154].

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 8900 ug/cu.m
MOE (Prov. Guid.) 1 hr. avg. 121000 ug/cu.m
MOE (Prov. Guid.) 24 hr. avg. 3000 ug/cu.m
Prop. 308 1 hr. avg. 73200 ug/cu.m

Advisories:

ACGIH TLV-TWA 100 ppm (360 mg/cu.m)
ACGIH TLV-STEL 150 ppm (540 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	7200.0000	ug/cu.m	8 hr	50.0
VA	6000.0000	ug/cu.m	24 hr	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 121 000 ug/cu.m.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.68 PROPYLENE GLYCOL MONOMETHYL ETHER ACETATE (108-65-6)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

PGMEA's systemic toxicity is similar to PGME, with the exception that PGMEA caused morphologic changes in the olfactory nasal mucosa of exposed rats and mice [154].

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 5000 ug/cu.m

MOE (Prov. Guid.) 24 hr. avg. 5000 ug/cu.m

Prop. 308 1 hr. avg. 4100 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found,

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 10000 ug/cu.m

Materials Damage:

No effects expected due to present ambient air concentrations.

C.69 PROPYLENE OXIDE (75-56-9)

Cancer Effects:

Classified as an A2 carcinogen by MAK (2). Massachusetts unit risk-- $6.67E-7$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

Propylene oxide is an irritant to the eyes, skin, respiratory passage and lungs [155]. Cases of corneal burns and contact dermatitis have also been reported. Central nervous system effects consist of incoordination, ataxia, and general depression.

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 13500 ug/cu.m

MOE (Prov. Guid.) 24 hr. avg. 4500 ug/cu.m

Prop. 308 24 hr. avg. 4500 ug/cu.m

Advisories:

MOL TWEAV 20 ppm (47 mg/cu.m)

ACGIH TLV-TWA 20 ppm (50 mg/cu.m)

OSHA PEL 240 mg/cu.m

IDLH 2000 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	0.0000			50.0
KS	0.0833	ug/cu.m	annual	0.0
MA	15.0000	ug/cu.m	24 hr.	0.0
NV	1.1900	mg/cu.m	8 hr.	42.0
NY	167.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	625.0000	ug/cu.m	1 yr.	420.0
VA	850.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.70 SELENIUM (7782-49-2)

Cancer Effects:

Conflicting data have been presented on the carcinogenic and teratogenic role of selenium. [156] notes that selenium is a suspected carcinogen and teratogen, but [157] states that selenium compounds have been shown to have anti-carcinogenic properties; particularly against cancers of the skin, liver, breast and colon. The EPA has not developed a cancer unit risk estimate for selenium.

Non-Cancer (Systemic) Effects:

Workers exposed to elemental selenium dusts have suffered catarrh, nose bleeds, and the loss of smell. Exposure to fumes has caused frontal headache, intense eye irritation, and difficulty breathing [156].

Standards:

Air:

MOE (Guideline) 0.5 hr. avg. 20 ug/cu.m

MOE (Guideline) 24 hr. avg. 10 ug/cu.m

Prop. 308 24 hr. avg. 10 ug/cu.m

Water:

MOE MAC 0.01 mg/L

MOE 0.01 mg/L for drinking water

MOE 0.02 mg/L for livestock and irrigation

Advisories:

MOL TWAEV 0.2 mg/cu.m

ACGIH TLV-TWA 0.2 mg/cu.m

IDLH 100 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	4.0000	ug/cu.m	8 hr	50.0
MA	0.2700	ug/cu.m	24 hr	0.0
MT	1.5700	ug/cu.m	24 hr.	128.0
	0.2600	ug/cu.m	annual	714.0
NV	0.0050	mg/cu.m	8 hr.	42.0
NY	0.6600	ug/cu.m	1 yr.	0.0
VA	3.5000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

Chronic plant toxicity limit is 0.2 ppm [157]. Studies have shown crop damage if Se exceeds 300 ppm. Severe foliar injury resulted from a pollution episode near Montreal involving a copper smelter. Peak airborne concentrations were 27.4 ug/cu.m, 5-day avg. was 15.4 ug/cu.m.

Aquatic Ecosystem Effects:

LC50's for freshwater fish range from 6.5 mg/L (rainbow trout) to 82 mg/L for fathead minnows. Freshwater macro-invertebrates are more sensitive. Lethal threshold for phytoplankton 2.5 ug/L [157].

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.71 SILICA

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

Occupational exposure to respirable silica causes silicosis. [158] reports 2 studies of workers exposed to respirable quartz. Workers exposed to less than 0.12 mg/cu.m did not develop silicosis. However, pottery workers exposed to 0.08 mg/cu.m developed silicosis.

Standards:

Air:

MOE (Guideline) 0.5 hr. avg. 15 ug/cu.m

MOE (Guideline) 24 hr. avg. 5 ug/cu.m

Prop. 308 24 hr. avg. 5 ug/cu.m

Advisories:

MOL TWEAV 0.10 mg/cu.m not to exceed 0.2 mg/cu.m

ACGIH TLV-TWA amorphous 10 mg/cu.m

ACGIH TLV-TWA cristobarite 0.05 mg/cu.m

ACGIH TLV-TWA quartz 0.1 mg/cu.m

ACGIH TLV-TWA fused 0.1 mg/cu.m

ACGIH TLV-TWA tridymite 0.05 mg/cu.m

ACGIH TLV-TWA tripoli 0.1 mg/cu.m

Ambient Air Levels:

No ambient levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.72 SODIUM CYANIDE (143-33-9)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

Sodium cyanide is super toxic, with a probable oral lethal dose in humans less than 5 mg/kg. The EPA developed an oral RfD of $4E-2$ mg/kg/day (140 ug/cu.m)a [159].

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 100 ug/cu.m

MOE (Prov. Guid.) 24 hr. avg. 100 ug/cu.m

Prop. 308 24 hr. avg. 35 ug/cu.m

Advisories:

OSHA PEL 5 mg/cu.m

IDLH 50 mg/cu.m

Ambient Air Levels:

No ambient air level found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.73 SODIUM HYDROXIDE (1310-73-2)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

Sodium hydroxide dusts are irritating to the upper respiratory system. Prolonged exposure to high concentrations may cause discomfort and ulceration of the nasal passage [160].

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 20 ug/cu.m

MOE (Prov. Guid.) 24 hr. avg. 10 ug/cu.m

Prop. 308 24 hr. avg. 10 ug/cu.m

Advisories:

ACGIH-C 2 mg/cu.m

Ambient Air Levels:

State	Conc. Unit	Avg. Time	Factor
CT	40.0000 ug/cu.m	8 hr	50.0
NV	0.0480 mg/cu.m	8 hr	42.0
SD	40.0000 ug/cu.m	8 hr.	50.0
VA	16.0000 ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

Corrosive.

C.74 STYRENE (100-42-5)

Cancer Effects:

Massachusetts unit risk- $5.70\text{E-}7$ ug/cu.m [180].

Non-Cancer (Systemic) Effects:

Styrene causes irritation of eyes, nose, and throat at approximately 200 ppm (852 mg/cu.m) [16]. At 376 ppm (1600 mg/cu.m) human volunteers reported eye, nose and skin irritation, nausea, headache, decreased co-ordination and a sense of inebriation [162]. Long-term occupational exposure have resulted in liver enlargement, disturbance of the liver's anti-toxic function, skin infection, reduced number of blood platelets and other blood disorders, and menstrual problems in female workers [162]. The EPA developed an oral RfD of $2\text{E-}1$ mg/kg/day (700 ug/cu.m) a [163]. EPA Baltimore IEMP TVs: Liver--699,3 ug/cu.m blood--699,3 ug/cu.m.

Standards:

Air:

MOE (standard) 0.5 hr. avg. 400 ug/cu.m
 MOE (standard) 24 hr. avg. 400 ug/cu.m
 Prop. 308 24 hr. avg. 400 ug/cu.m

Advisories:

MOL TWEAV 50 ppm (213 mg/cu.m)
 MOL STEV 200 ppm (852 mg/cu.m)
 ACGIH TLV-TWA 50 ppm (215 mg/cu.m)
 ACGIH TLV-STEL 100 ppm (425 mg/cu.m)
 OSHA PEL 100 ppm.

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	4300.0000	ug/cu.m	8 hr	50.0
IN	3.4483	ug/cu.m	annual	0.0
KS	34.4800	ug/cu.m	annual	0.0
MA	39.0000	ug/cu.m	24 hr	0.0
NC	42500.0000	ug/cu.m	15 min	10.0
NV	5.1190	ug/cu.m	8 hr	42.0
NY	716.0000	ug/cu.m	1 yr	0.0
RI	30.0000	ug/cu.m	annual	0.0
VA	3600.0000	ug/cu.m	24 hr	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

Freshwater fish LC50's 25.05 mg/L-74.83 mg/L

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Perception 0.036 mg/cu.m [164]

Odor threshold 1 ppm (4.26 mg/cu.m)

Odor threshold 4.7E-2 ppm (0.2 mg/cu.m) [145].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.75 SULPHUR DIOXIDE (A) (7446-09-5)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

MORTALITY: (Deaths/100,000) Lower Bound = 0 Central Case = 2.672
* (change in SO₂ ug/cu.m). Upper Case = 4 * (change in SO₂
ug/cu.m). Value: \$4,500,000 (1986, U.S.).

HOSPITAL DAYS FOR RESPIRATORY CONDITIONS: Lower Bound = 0.00015
* (change in SO₂ ug/cu.m) Central Case = 0.0004 * (change in SO₂
ug/cu.m) Upper Case = 0.00066 * (change in SO₂ ug/cu.m). Value:
\$1050/day (1987, U.S.).

HOSPITAL ADMISSIONS FOR RESPIRATORY DISEASE: Lower Bound = 0.63
* (change in SO₂ ug/cu.m) Central Case = 4.065 * (change in SO₂
ug/cu.m) Upper Case = 7.5 * (change in SO₂ ug/cu.m). Value:
\$6500 (1987, U.S.).

Standards:

Air:

MOE (standard) 0.5 hr. avg. 830 ug/cu.m
MOE (Standard) 24 hr. avg. 275 ug/cu.m
MOE (Standard) 1 hr. avg. 690 ug/cu.m
MOE (Standard) 1 yr. 55 ug/cu.m
Prop. 308 24 hr. avg. 275 ug/cu.m, 1 hr. avg.
690 ug/cu.m, 1 yr. avg. 55 ug/cu.m (mean)

Advisories:

MOL TWEAV 2ppm (5.2/cu.m)
MOL STEV 5 ppm (10.4 mg/cu.m)
ACGIH TLV-TWA 2 ppm (5 mg/cu.m)
ACGIH TLV-STEL 5 ppm (10 mg/cu.m)
OSHA PEL 13 mg/cu.m
EEGL/SPEGL 10 ppm
IDLH 100 ppm

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
AZ-Pigico	80.0000	ug/cu.m	annual	0.0
	365.0000	ug/cu.m	24 hr	0.0
	1300.0000	ug/cu.m	3 hr.	0.0
CT	860.0000	ug/cu.m	8 hr.	50.0
NV	0.1190	mg/cu.m	8 hr.	42.0
TN-Chat.	1.2000	ug/cu.m	1 yr.	0.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

Phytotoxicity threshold based on Linzon (1978) set at 0.02 ppm (52 ug/cu.m) for annual average [165].

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold $4.7E-1$ ppm (1.2 mg/cu.m) [145].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.76 SULFURIC ACID (7664-93-9)

Cancer Effects:

Not considered carcinogenic by the ACGIH [166].

Non-Cancer (Systemic) Effects:

Chronically exposed workers may show various lesions of the skin, tracheo bronchitis, stomatitis, conjunctivitis, or gastritis. Tooth enamel erosion has also been reported [166].

Standards:

Air:

Prop. 308 24 hr. avg. 35 ug/cu.m.

Advisories:

MOL TWEAV 1 mg/cu.m
ACGIH TLV-TWA 1 mg/cu.m
ACGIH TLV-STEL 3 mg/cu.m
OSHA PEL 1 mg/cu.m
EEGL/SPEGL 1 mg/cu.m
IDLH 80 mg/cu.m

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	20.0000	ug/cu.m	8 hr.	50.0
KS	2.3810	ug/cu.m	annual	420.0
MA	14.0000	ug/cu.m	24 hr.	0.0
NV	0.0240	mg/cu.m	8 hr.	42.0
VA	16.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

Strongly corrosive.

C.77 SUSPENDED PARTICULATE MATTER LESS THAN 44 UM [A]

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

MORTALITY: $Y = \text{change in annual deaths}/100,000$. Lower Bound: $Y = 0$ Central Case: $Y = .221 * \text{change in TSP}$ Upper Bound: $Y = .625 * \text{change in TSP}$. Where TSP = annual arithmetic mean in ug/cu.m. Value: Lower: \$1,000,000; Central: \$4,500,000.

EMERGENCY ROOM VISITS (ERV): $Y = \text{change in annual per capita ERV}$. Lower Bound: $Y = .000012 * \text{change in TSP}$ Best Case: $Y = .00013 * \text{change in TSP}$ Upper Case $Y = .00025 * \text{change in TSP}$. Where TSP = annual arithmetic mean in ug/cu.m. Value: Central: \$175.

RESTRICTED ACTIVITY DAYS (RAD): $Y = \text{change in RAD per year per person}$. Lower Bound: $Y = (.01938 * \text{TSP change}) - \text{ERV change}$ Central Case: $Y = (.02736 * \text{TSP change}) - \text{ERV change}$ Upper Bound: $Y = (.04334 * \text{TSP change}) - \text{ERV change}$. Where TSP is annual arithmetic mean in ug/cu.m. Value: High: \$52 U.S. 1986; Central: \$44; Low: \$12.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE: RR = relative risk of bronchitis in adults: $RR = .992 + .00004755 * T$. Where T = ug/cu.m hours of TSP per year. Y = change in probability of bronchitis in children under 16 years old: $Y = .001096 * T$. Where T = change in 1 yr. annual average exposure to TSP.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 100 ug/cu.m

MOE (Standard) 24 hr. avg. 120 ug/cu.m

MOE (Standard) 1 yr. 60 ug/cu.m

Prop. 308 24 hr. avg. 120 ug/cu.m, 1 yr. avg. 60 mg/cu.m (geo. mean).

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

Standard set to protect visibility. Y = change in visibility values per household in \$1986 U.S. Lower Bound: $Y = 103 * \ln(V2/V1)$ Central Case: $Y = 206 * \ln(V2/V1)$ Upper Bound: $Y = 824 * \ln(V2/V1)$. Where: $V2$ = new visibility after change (visual range in miles) $V1$ = old visibility prior to change (in miles) VR = visual range in miles $VR = 605/FP$ Where FP is matter, 2.5 microns. This can be converted to TSP by $FP = b * TSP$. In U.S. cities, b ranges from about 0.25 to 0.45.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

Y = change in annual household damage U.S. \$1986 Lower Bound: $Y = 0.52 * \text{change in TSP}$ Best Case: $Y = 7.4 * \text{change in TSP}$ Upper Bound: $Y = 14.8 * \text{change in TSP}$.

C.78 TETRACHLOROBENZENES (95-94-3)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

The EPA developed an oral RfD of $3\text{E-}4$ mg/kg/day (1 ug/cu.m)a [167].

Standards:

Air:

Unable to locate MOE air quality guideline or regulation.

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air level found.

Animal Effects:

Oral LD 50s range from 1035 mg/kg (mouse) to 3105 mg/kg (rat) [168].

Phytotoxic Effects:

Decrease in seedling germination and growth in soils treated with 1.9 kg- 151.9 kg/ha [168].

Aquatic Ecosystem Effects:

Freshwater fish LC50s 1.55 mg/L- 5.69 mg/L Chronic NOELS 0.32 mg/L- 0.68 mg/L [168].

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.79 TETRACHLOROPHENOLS (58-90-2)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

2,3,4,6 is highly toxic by ingestion and inhalation. Dust may irritate nose and pharynx [169]. The EPA has developed an oral RfD of $3E-2$ mg/kg/day (105 ug/cu.m)a [170].

Standards:

Unable to locate MOE air quality guideline or regulation.

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.80 2,3,7,8-TETRACHLORO-DIBENZO-P-DIOXIN (1746-01-6)

Cancer Effects:

2,3,7,8-TCDD has been found to be carcinogenic in rats and mice. It has not been possible to predict the ability of 2,3,7,8-TCDD to cause tumors in humans [171]. The U.S. EPA has not published quantitative risk estimates of the carcinogenic potential of 2,3,7,8-TCDD within IRIS. Classified as a 2B carcinogen by IARC, b by NTP, and as a carcinogen by NIOSH [2].

Non-Cancer (Systemic) Effects:

Acute exposure to PCDDs or PCDFs results in symptoms which slowly decrease over a prolonged period of time. Accidents at chemical plants in Italy and England have linked TCDD exposure to skin lesions and chloracne. In almost all cases, chloracne disappeared after a few years. Current evidence is insufficient to establish a causal relationship between exposure to PCDDs or PCDFs and chronic health effects relating to systemic effects such as coronary disease or impairment of the immune system [171]. The MOE estimated an ADI for 2,3,7,8-TCDD of $1\text{E}-5$ ug/kg/day for humans.

Standards:

Prop. 308 1 yr. avg. $x/30 + y/30$ (50) = 1 where x = concentration (pg/cu.m) CDDs and y = concentration (pg/cu.m) CDFs.

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.81 THIOUREA (62-56-6)

Cancer Effects:

IARC considers thiourea to be an animal carcinogen; there is no information available to assess its carcinogenicity in man [172]. Classified as a 3 carcinogen by IARC, and b by NTP [2].

Non-Cancer (Systemic) Effects:

Limited acute and chronic toxicity data are available. Chronic exposure may cause agranulocytosis.

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 60 ug/cu.m

MOE 24 hr. avg. 20 ug/cu.m

Prop. 308 24 hr. avg. 20 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.82 TIN (7440-31-5)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

Tin salts are highly toxic after they have gained access to the blood stream, causing paralysis and other neurologic damage [173]. Organic tin compounds have been linked to skin irritation and effects on the central nervous system and circulatory system [174].

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 30 ug/cu.m

MOE 24 hr. avg. 10 ug/cu.m

Prop. 308 24 hr. avg. 10 ug/cu.m

Advisories:

MOL TWEAV 2 mg/cu.m for metal, oxide and inorganic compounds

MOL TWEAV 0.1 mg/cu.m organic Sn cmpds

ACGIH TLV-TWA 2 mg/cu.m

ACGIH TLV-TWA 0.1 mg/cu.m organic Sn compounds

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	2.0000	ug/cu.m	8 hr.	50.0
NV	0.0480	mg/cu.m	8 hr.	42.0
VA	1.6000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.83 TOLUENE (108-88-3)

Cancer Effects:

Does not appear to be carcinogenic [175].

Non-Cancer (Systemic) Effects:

Acute exposure may cause mental confusion, dizziness and inappropriate behavior. 200 ppm (752 mg/cu.m) for 7 hours caused mild eye irritation; 600 ppm (2256 mg/cu.m) for 7 hours lassitude, nausea, and hilarity; 800 ppm (3008 mg/cu.m) for 7 hours metallic taste in mouth and transitory headache, extreme lassitude, inebriation, and slight nausea. Chronic exposure results in neurotoxicity and kidney and liver pathology [175]. The EPA developed an oral RfD of $3E-1$ mg/kg/day (1050 ug/cu.m)a [176]. EPA Baltimore IEMP TVs: liver--1050.0 ug/cu.m kidney--1050.0 ug/cu.m reproductive--500 ug/cu.m blood--1050 ug/cu.m neurobehavioral--580.0 ug/cu.m respiratory--1010 ug/cu.m fetal/developmental--476.0 ug/cu.m.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 2000 ug/cu.m

MOE (Standard) 24 hr. avg. 2000 ug/cu.m

Prop. 308 24 hr. avg 2000 ug/cu.m

Advisories:

MOL TWEAV 100 ppm (376 mg/cu.m)

MOL STEV 150 ppm (564 mg/cu.m)

ACGIH TLV-TWA 100 ppm (375 mg/cu.m)

ACGIH TLV-STEL 150 ppm (560 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
AZ	1.0000	ppm	1 hr.	100.0
CT	7500.0000	ug/cu.m	8 hr.	50.0
IN	1875.0000	ug/cu.m	8 hr.	200.0
MA	51.0000	ug/cu.m	24 hr.	0.0
NC	56000.0000	ug/cu.m	15 min.	10.0
NV	8.9290	mg/cu.m	8 hr.	42.0
NY	7500.0000	ug/cu.m	1 yr.	0.0
RI	2000.0000	ug/cu.m	24 hr.	0.0
SD	7500.0000	ug/cu.m	8 hr.	50.0
VA	6000.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

0.17 mg/cu.m Odor threshold 4.68 ppm (17.6 mg/cu.m)
Odor threshold 2.14 ppm (8 mg/cu.m) [145].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.84 TOLUENE DIISOCYANATE (584-84-9)

Cancer Effects:

Massachusetts unit risk-- $6.79\text{E-}6$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

The major effects of toluene diisocyanate is in the respiratory tract. It also produces inflammation and sensitization of the skin, lacrimation, smarting, burning in the eyes, abdominal distress, nausea, and vomiting [177]. Acute exposure to vapor can produce severe irritant effects on mucous membranes, the respiratory tract, and the eyes. Chronic exposure may result in coughing, wheezing, and shortness of breath [177].

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 1 ug/cu.m

MOE (Standard) 24 hr. avg. 0.5 ug/cu.m

Prop. 308 24 hr. avg. 0.5 ug/cu.m

Advisories:

MOL TWEAV 0.005 ppm (0.2 mmoles/cu.m)

MOL STEV 0.02 ppm (0.8 mmoles/cu.m)

ACGIH TLV-TWA 0.005 ppm (0.04 mg/cu.m)

ACGIH TLV-STEL 0.02 ppm (0.15 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
MA	0.4800	ug/cu.m	24 hr.	0.0
SD	0.7200	ug/cu.m	8 hr.	50.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Perception 0.020 mg/cu.m [178].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.85 TOTAL REDUCED SULFUR

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

No IRIS, ACGIH, EPA or MOE information available.

Standards:

Air:

MOE (Prov. Guid.) 0.5 hr. avg. 40 ug/cu.m

MOE (Prov. Guid.) 1 hr. avg. 40 ug/cu.m

Prop. 308 1 hr. avg. 40 ug/cu.m

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air level found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.86 1,2,4-TRICHLOROBENZENE (120-82-1)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

Systemic toxicity occurs in rats below 25 ppm (200 mg/cu.m). sublethal doses have caused liver damage to guinea pigs [179]. Irritation occurs at approximately 5 ppm (40 mg/cu.m) in the workplace. No pathological skin changes occur [180]. The EPA has developed an oral RfD of $2E-2$ mg/kg/day (70 ug/cu.m)a [181].

Standards:

Air:

MOE (Guideline) 0.5 hr. avg. 100 ug/cu.m

MOE (Guideline) 24 hr. avg. 400 ug/cu.m

Prop. 308 24 hr. avg. 35 ug/cu.m

Advisories:

ACGIH TLV-C 5 ppm (40 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	800.0000	ug/cu.m	8 hr.	50.0
NV	0.9520	mg/cu.m	8 hr.	42.0
NY	133.0000	ug/cu.m	1 yr.	0.0
VA	350.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 3 ppm (24 mg/cu.m) [179].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.87 1,1,1-TRICHLOROETHANE (71-55-6)

Cancer Effects:

Suggested human carcinogen. Massachusetts unit risk-- 1.60×10^{-5} ug/cu.m [18].

Non-Cancer (Systemic) Effects:

The EPA has developed an oral RfD of 9×10^{-2} mg/kg/day (315 ug/cu.m)a [182].

Standards:

Prop. 308 24 hr. avg. 115000 ug/cu.m

Advisories:

MOL TWEAV 350 ppm (1910 mg/cu.m)
MOL STEV 450 ppm (2455 mg/cu.m)
ACGIH TLV-TWA 350 ppm (1900 mg/cu.m)
ACGIH STEL 450 ppm (2450 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	38000.0000	ug/cu.m	8 hr.	50.0
IN	19000.0000	ug/cu.m	8 hr.	200.0
MA	1300.0000	ug/cu.m	24 hr.	0.0
N C	9500.0000	ug/cu.m	24 hr.	200.0
NV	45.2380	mg/cu.m	8 hr.	42.0
NY	38000.0000	ug/cu.m	1 yr.	0.0
SD	38000.0000	ug/cu.m	8 hr.	50.0
VA	32000.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.88 1,3,5-TRICHLOROBENZENE (108-70-3)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

Moderately irritating to the skin and has been associated with liver injury and hair loss in humans [183].

Standards:

Unable to locate MOE air quality guideline or regulation.

Advisories:

No workplace standards found.

Ambient Air Levels:

No ambient air levels found.

Animal Effects:

No effects expected due to present ambient air concentrations.

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

No effects expected due to present ambient air concentrations.

Materials Damage:

No effects expected due to present ambient air concentrations.

C.89 TRICHLOROETHYLENE (79-01-6)

Cancer Effects:

Classified as a probable human carcinogen by the EPA on the basis of positive responses in 2 strains of mice by 2 routes and suggestive increases in tumor incidences in male rats by gavage [184]. IRIS inhalation slope factor-- $1.3\text{E-}2$ mg/kg/day IRIS inhalation unit risk-- $1.3\text{E-}6$ ug/cu.m Classified as a B carcinogen by MAK and as a carcinogen by NIOSH [2]. Not classified as a carcinogen by the ACGIH [185]. Massachusetts unit risk-- $1.63\text{E-}6$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

Principal targets for inhaled trichloroethylene are the central nervous system, liver, kidney, and hematological system [186]. Exposure to 27 ppm (145 mg/cu.m) has caused drowsiness and mucous membrane irritation. Long term exposures to workers have caused vertigo, headache, and short term memory loss [186]. ATSDR [186] estimated MRLs for trichloroethylene inhalation are: acute--0.2 ppm (1.07 mg/cu.m) intermediate--0.4 ppm (2.14 mg/cu.m) EPA Baltimore IEMP TVs: kidney--3770.0 ug/cu.m liver--25.9 ug/cu.m neurobehavioral--25.9 ug/cu.m.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 85000 ug/cu.m
MOE (Standard) 24 hr. avg. 28000 ug/cu.m
Prop. 308 24 hr. avg. 28000 ug/cu.m

Advisories:

MOL TWAEV 50 ppm (268 mg/cu.m)
MOL STEV 200 ppm (1075 mg/cu.m)
ACGIH TLV-TWA 50 ppm (270 mg/cu.m)
ACGIH TLV-STEL 200 ppm (1080 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
AZ	0.0500	ppb	70 yrs.	0.0
CT	1350.0000	ug/cu.m	8 hr.	200.0
IN	2675.0000	ug/cu.m	8 hr.	200.0
KS	2.4390	ug/cu.m	annual	0.0
MA	6.1000	ug/cu.m	24 hr.	0.0
NC	108.0000	mg/cu.m	15 min.	10.0
NV	6.4290	mg/cu.m	8 hr.	42.0
NY	900.0000	ug/cu.m	1 yr.	0.0
PA-Phil.	6840.0000	ug/cu.m	1 yr.	42.0
SD	2700.0000	ug/cu.m	8 hr.	100.0
VA	4500.0000	ug/cu.m	24 hr.	60.0

Animal Effects:

Oral LD50s from 2402 to 7200 mg/kg [184].

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

Fish 96 hr. LC50s range between 100-1000 ppm [184].

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

Odor threshold 580 mg/cu.m [184].

Odor threshold 2.14 ppm (11.5 mg/cu.m) [187].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.90 2,4,6-TRICHLOROPHENOL (88-06-2)

Cancer Effects:

Classified as a probable human carcinogen by the EPA on the basis of increased incidence of lymphomas or leukemias in male rats and hepatocellular adenomas or carcinomas in male and female mice [188]. Classified as a 2B carcinogen by IARC and b by NTP [2]. IRIS inhalation slope factor-- $2E-2$ mg/kg/day IRIS inhalation unit risk-- $5.7E-6$ ug/cu.m Massachusetts unit risk-- $6.20E-6$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

No IRIS, ACGIH, EPA or MOE information available.

Standards:

Unable to locate MOE air quality guideline or regulation.

Advisories:

No workplace standards found.

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
MA	0.0000	ug/cu.m	24 hr.	0.0

Animal Effects:

Oral LD50 (rat) 820-4000 mg/kg [189].

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

0.011 mg/L @ 30 C

0.333 mg/L @ 60 C [189].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.91 VINYL CHLORIDE (75-01-4)

Cancer Effects:

Inhalation of vinyl chloride is carcinogenic in rats, mice, and hamsters [190]. It has been associated with cancers of the liver, brain, lung, and possibly other sites due to occupational exposures [190]. The EPA has not developed a cancer unit risk factor for vinyl chloride. Classified as a 1 carcinogen by IARC, A1 by MAK, a by NTP, A1 by ACGIH, and as a carcinogen by NIOSH [2]. Massachusetts unit risk-- $2.60\text{E-}6$ ug/cu.m [18].

Non-Cancer (Systemic) Effects:

Vinyl chloride is considered to have very low toxicity via acute inhalation. However, short-term acute exposures may cause dizziness, headache, unconsciousness and death. Long-term chronic workplace exposures have resulted in damage to the liver, lungs, reduced circulation in the fingers, thickening of the skin, and changes in the blood [192]. ATSDR estimated MRLs for vinyl chloride inhalation are: acute--0.8 ppm (2.08 mg/cu.m) intermediate--0.08 ppm (0.208 mg/cu.m) EPA Baltimore IEMP TV: liver--4.5 ug/cu.m.

Standards:

Air:

MOE (Guideline) 0.5 hr. avg. 560 ug/cu.m

MOE (Guideline) 24 hr. avg. 280 ug/cu.m

Prop. 308 24 hr. avg. 280 ug/cu.m

Advisories:

MOL TWEAV 2 ppm (5.2 mg/cu.m)

MOL STEV 10 ppm (26 mg/cu.m)

ACGIH TLV-TWA 5 ppm (10 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CT	50.0000	ug/cu.m	8 hr.	200.0
IL	0.0000			0.0
KS	3.8460	ug/cu.m	annual	0.0
MA	3.9000	ug/cu.m	24 hr.	0.0
NC	50.0000	ug/cu.m	24 hr.	200.0
NV	0.2380	mg/cu.m	8 hr.	42.0
NY	0.4000	ug/cu.m	1 yr.	0.0
PA-Phil.	2.5700	ppb	1 yr.	420.0
SD	50.0000	ug/cu.m	8 hr.	200.0
VA	1.0000	ug/cu.m	24 hr.	100.0

Animal Effects:

Rat oral LC50 390 mg/L [191].

Phytotoxic Effects:

No effects expected due to present ambient air concentrations.

Aquatic Ecosystem Effects:

No effects expected due to present ambient air concentrations.

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

3000 ppm (7800 mg/cu.m) [191].

TOC 26-52 mg/cu.m [192].

Materials Damage:

No effects expected due to present ambient air concentrations.

C.92 XYLENES (1330-20-7)

Cancer Effects:

Not considered carcinogenic.

Non-Cancer (Systemic) Effects:

200 ppm (870 mg/cu.m) causes irritation to eyes, nose, and throat. Higher concentrations may cause dizziness, nausea, and unconsciousness. Change in heart rhythms as well as reversible damage to kidneys and liver have been reported [193]. The EPA oral RfD for xylenes (mixed isomers) is 2E+0 mg/kg/day (7000 ug/cu.m)a [194]. EPA Baltimore IEMP TVs: liver--215.0 ug/cu.m neurobehavioral--215.0 ug/cu.m respiratory--215.0 ug/cu.m cardiovascular--215.0 ug/cu.m blood--215.0 ug/cu.m kidney--215.0 ug/cu.m reproductive--52.8 ug/cu.m fetal/developmental--52.8 ug/cu.m.

Standards:

Air:

MOE (Standard) 0.5 hr. avg. 2300 ug/cu.m

MOE (Standard) 24 hr. avg. 2300 ug/cu.m

Prop. 308 24 hr. avg. 2300 ug/cu.m

Advisories:

MOL TWEAV 100 ppm (435 mg/cu.m)

MOL STEV 150 ppm (650 mg/cu.m)

ACGIH TLV-TWA 100 ppm (435 mg/cu.m)

ACGIH TLV-STEL 150 ppm (655 mg/cu.m)

Ambient Air Levels:

State	Conc.	Unit	Avg. Time	Factor
CO-ElPso	0.0000			0.0
CT	8680.0000	ug/cu.m	8 hr.	50.0
IN	2175.0000	ug/cu.m	8 hr.	200.0
MA	59.2000	ug/cu.m	24 hr.	0.0
NC	65500.0000	ug/cu.m	15 min.	10.0
NV	10.3750	mg/cu.m	8 hr.	42.0
NY	1450.0000	ug/cu.m	1 yr.	0.0
RI	700.0000	ug/cu.m	24 hr.	0.0
SD	8700.0000	ug/cu.m	8 hr.	50.0

Animal Effects:

Rat inhalation LC50 47.635 mg/cu.m [193].

Phytotoxic Effects:

Young barley and tomato plants injured when exposed to xylene vapor [193].

Aquatic Ecosystem Effects:

M-Xylene freshwater fish LC50 (low) 16 mg/L [195].

Surface Water Effects:

No effects expected due to present ambient air concentrations.

Effects on Visibility:

No effects expected due to present ambient air concentrations.

Odor Effects:

0.3-1.0 ppm (1.3-4.35 mg/cu.m) in water [195].

Materials Damage:

No effects expected due to present ambient air concentrations.

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